# A COMPREHENSIVE STUDY ON ILEAL PERFORATION

Dissertation submitted to

The Tamil Nadu Dr. M.G.R. Medical University,

Chennai – 600032

*With partial fulfillment of the regulations*

*for the award of Degree*

M.S. GENERAL SURGERY

BRANCH – I


**DEPARTMENT OF SURGERY,**

**K.A.P.V. GOVT. MEDICAL COLLEGE, TRICHY.**

**THE TAMIL NADU DR. M.G.R. MEDICAL UNIVERSITY,**

**CHENNAI, INDIA**

**APRIL 2013**

# CERTIFICATE

This is to certify that this dissertation titled

"**A COMPREHENSIVE STUDY ON ILEAL PERFORATION**"

is a bonafide work of **Dr.Arunan.T**., Post Graduate in M.S. General Surgery, Department of General Surgery, K.A.P.V. Government Medical College, Trichy and has been prepared by him under our guidance. This has been submitted in partial fulfillment of regulations of The Tamil Nadu Dr. M.G.R. Medical University, Chennai -32 for the award of M.S. Degree in General Surgery (Branch- I).

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# DECLARATION

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I thank **Prof. Dr.A. Kanakasundarram,M.S.,** Head of Department, General Surgery, for helping me and guiding me during this study.

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Last but not the least, I gratefully thank the patients without whom this study could not been completed.

**Dr.ARUNAN.T**

## ABSTRACT

**Background & Objectives**: Ileal perforation is one of the commonest occurrence in our hospital setup, with the majority of cases having an etiology of trauma. The aim is to study the various causes of ileal perforation and its presentation and various surgical procedure and its complications and factors affecting the outcome.

**Methods**: Seventy cases of ileal perforation were included in this study from the period between October 2010 and October 2012. Factors were tabulated and statistically analyzed to study their contribution.

**Results:** Trauma was the most common cause of ileal perforation in this study followed by non-specific perforations. Patients presented primarily in the third and fourth decades of life with a male preponderance. Many patients had air under diaphragm in X-rays and underwent surgery within 24hrs of onset . 70% of patients underwent 2 layer closure with complication rate of 67% and mortality rate of 5.7% .

**Conclusion**: We found trauma as the most common etiology for ileal perforation. Incidence of typhoid induced perforations seems to have significantly reduced. Fecal peritonitis, age , shock, lag period were found to be significant in contributing to mortality and morbidity.

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# *INTRODUCTION*

# INTRODUCTION

Ileal perforation is one of the commonest problem seen in tropical countries. The commonest cause being trauma. In western countries the causes are malignancy, trauma and mechanical etiology, in the order of frequency (1,2,3).

Over the years a definite changing trend was observed in ileal perforations both in terms of causes, treatment and prognosis.Early and Better antibiotics, aggressive surgery and the elimination of conservative treatment, good preoperative and postoperative care have all significantly contributed to the improvement in patient outcome (4,5).

It is true that outcomes were improved but still cases of ileal perforation cause a significant morbidity and mortality that persists despite of significant changes in health care over the years .

Our dissertation aims to study the etiology, presentation, management outcome and the factors influencing prognosis and outcome in ileal perforations The present study includes 70 patients of ileal perforation with emphasis on trauma, nonspecific and tubercular and typhoid perforations and the factors influencing outcome.

# *AIM AND OBJECTIVES*

# AIM AND OBJECTIVES

The aims and objectives of this study are

- To study the various causes of ileal perforation.
- To study the presentation of ileal perforation.
- To study the different modes of surgical management of patients admitted with ileal perforation.
- To study the various complications and outcome of these patients.
- To study the factors influencing the outcome of these patients.

# *REVIEW OF LITERATURE*

# REVIEW OF LITERATURE

## HISTORICAL REVIEW

Ileal perforation is one of the commonest surgical emergency encountered in a day to day practice. Its history goes back to the period of SUSHRUTHA where perforation of intestines by sharp objects, fish bones and thorns has been described. There was also available a record of perforated intestines being drawn out and the severed edges were held close by the large black ants with their jaws prior to the clipping of the bodies of ants ,following which the bowel was put inside and abdomen closed[(6)].

In 1776 William Cullen was the one to coin the term PERITONITIS[(7)]. The first successful closure of perforated intestine was performed by BENJAMIN TRAVERS[(8)]. The introduction of LEMPERT SUTURES[(7)] had begun a new era in restoring the perforated bowel integrity.

The term TYPHUS (gr.cloudy) was first used by Hippocrates[(9)] in 460 B.C. The term typhoidea was first used by Louis in 1829 and about 150 cases with splenomegaly, rose spots[(10)], fever,intestinal perforation, cervical lymphadenopathy were described by him. In 1973 Budd explained a theory of faeco oral transmission of the diease.

1880 - Discovery of typhoid bacilli by Karl Joseph Ebereth[10]

1884 - Isolation & culture of Salmonella typhi by Gaffkey[10]

1896 - Test for agglutination detection in serum of typhoid patients by Widal .

First typhoid vaccine in humans was introduced by Pfeiffer and Kalle [10] .Before reviewing the aetiology , first we will try to summarise briefly the anatomy of ileum which helps us in understanding the later pages .

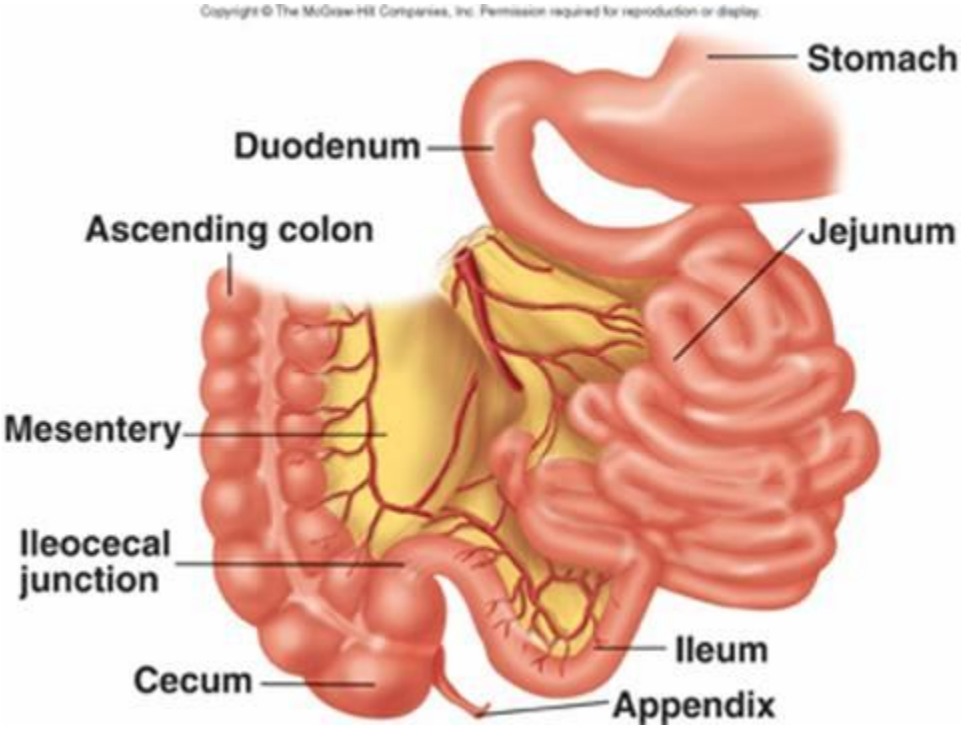
**ANATOMY OF ILEUM**

The small intestine is a tubular structure extending from the pylorus to the cecum and the estimated length of this structure is thought to measure about 4 to 6 m in the living[72]. It consists of three segments in series to each other : the duodenum, jejunum, and ileum. The duodenum, the most proximal part, lies partly in the retroperitoneum and adjacent to the head and inferior border of the body of the pancreas. It is being demarcated from the stomach by the pyloric antrum and from the jejunum by the ligament of Treitz.

The jejunum and ileum are intraperitoneal and are attached to the retroperitoneum by a broad-based mesentery. No anatomic landmark distinctly demarcates the jejunum from the ileum; the proximal 40% of the

jejunoileal segment is arbitrarily defined as the jejunum and the distal 60% as the ileum. The ileum is demarcated from the cecum by the ileocecal valve[72].

The jejunum and ileum are being suspended from the posterior portion of the peritoneal cavity by a long mesentery that has a oblique travel from the left upper quadrant to the right lower quadrant of the abdomen[71] .

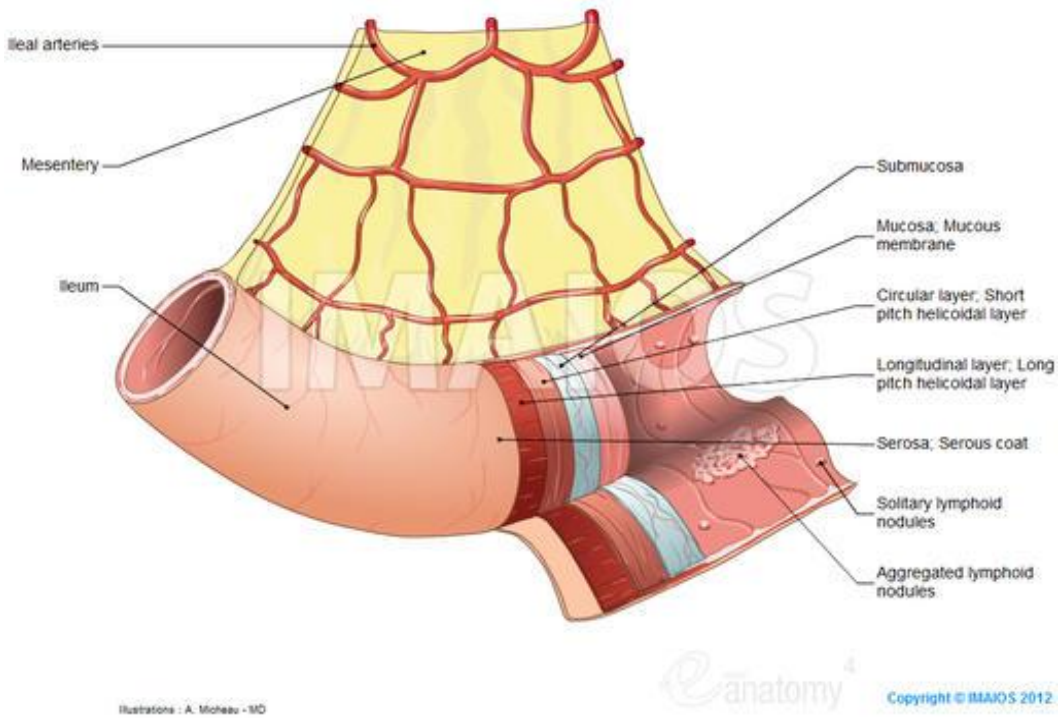


Features differentiating jejunum from ileum are as follows [71,72,73]

| FEATURE | JEJUNUM | ILEUM |
|---|---|---|
| Vasa recta | Longer , straight | Shorter with more arterial arcades |
| Diameter | Larger | Smaller |
| Colour | Pink | Pale |
| Wall thickness | Thicker | Thinner |
| Fatty mesentery | Less | More |
| Speciality | Plica circularis or valvulae conniventes | Paeyer's patches |

**BLOOD SUPPLY[71]**

The blood supply, as well as fat and lymphatics, resides within the Mesentry.The blood supply of the jejunum and ileum is derived from the Superior mesenteric artery, which has an extensive anastomosis (the vasa recta) adjacent to the mesenteric border of the bowel named the marginal artery. This artery travels along the entire length of the bowel. Near the ileal branch of the ileocolic artery, this artery breaks up into the anastomotic network of the vasa recta which increases in its complexity. Venous drainage follows the course of the arteries .




## LYMPHATIC DRAINAGE

Lymphatic drainage occurs through the lymphatic vessels travelling parallel to the corresponding arteries. This lymph drains through the mesenteric lymph nodes into the cisterna chyli, then through the thoracic duct, and ultimately drains into the left subclavian vein [(72)].

This lymphatic drainage constitutes a major route for the transport of absorbed lipid into the systemic circulation and likewise plays a major pathway in immune defense mechanism and also in the spread of metastasis of tumor cells from the carcinomas of small bowel [(73)].

## NERVE SUPPLY

The innervation is provided by both sympathetic and parasympathetic divisions of the autonomic nervous system . Parasympathetic innervation is derived from the vagus . They traverse the celiac ganglion and synapse in the submucosal (Meissner's) and myenteric (Auerbach's) plexus.The myenteric plexus is responsible for the basal electrical activity of the gut. Stimulation of the parasympathetic system prepares for the activity of intestine by increasing blood flow, contractility, and its secretions.

The sympathetic fibers are from three sets of splanchnic nerves and their ganglion cells being situated usually in a plexus which is around the base of the superior mesenteric artery. Motor impulses affect the gut secretion and motility. Afferents from the intestine is carried through the general visceral afferent fibers of the sympathetic nervous system.

## HISTOLOGY

The wall of small intestine is made up of 4 distinct layers ,

- Mucosa
- Submucosa
- Muscularis propria
- Serosa

## SEROSA

It is the outermost layer of the small intestine consisting of visceral peritoneum, which is a single layer of flattened mesoepithelial cells encircling the jejunoileum, and anterior surface of the duodenum.

## MUSCULARIS PROPRIA

The muscularis propria has two muscle layers, an outer thin longitudinal layer, and a inner thicker circular layer of smooth muscles. Ganglion cells from the myenteric (Auerbach) plexus are being scattered between these two muscle layers and send nerve fibers into both layers, thus permitting conduction through the muscle layer[72].

## SUBMUCOSA

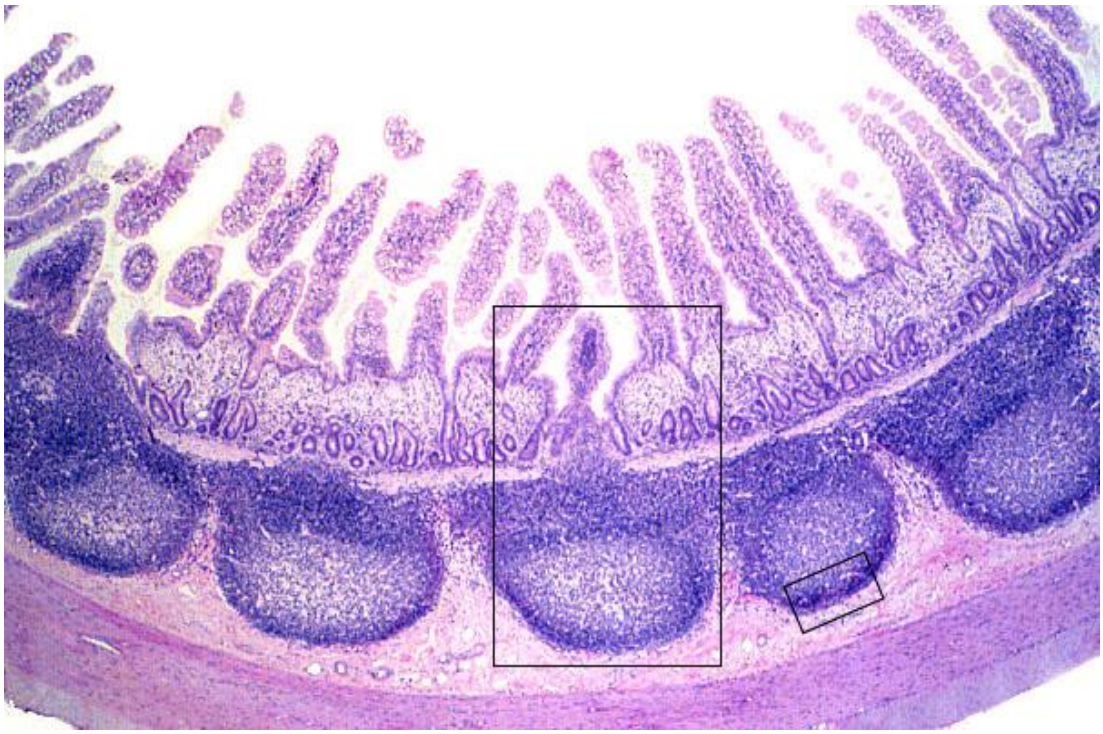
It has a layer of fibroelastic connective tissue which contains blood vessels and nerve fibres. It is the strongest component of the bowel wall and therefore should be included in anastomotic sutures [73]. It contains extensive networks of lymphatics, arterioles, and venules and plexus of nerve fibers and ganglion cells (Meissner plexus). The nerve fibres from the mucosa & submucosa muscle layers are interconnected by small nerves, and cross connections between cholinergic and adrenergic elements have been evaluated .

MUCOSA

It is divided into three layers:

- Muscularis mucosa
- Lamina propria
- Epithelial layer

The muscularis mucosa is a thin muscle layer separating the mucosa from the submucosa.The lamina propria is a layer of connective tissue between the epithelium and the muscularis mucosa containing plasma cells, mast cells, eosinophils ,lymphocytes,macrophages, fibroblasts, smooth muscle cells, and noncellular connective tissue. These plasma cells synthesize immunoglobulins. Other immune cells in the lamina propria release various inflammatory mediators (e.g., cytokines,histamines and arachidonic acid metabolites) that modulates various cellular functions of the overlying epithelium. The epithelial layer is a continuous sheet of epithelial cells covering the villi and the crypts. The functions of the crypt epithelium are cell renewal and endocrine,exocrine, water, and ion secretion . The functions of the villus epithelium are absorption and digestion.



Four main cell types are found in the mucosal layer:

- Goblet cells secreting mucus
- Paneth cells secreting lysozyme, tumor necrosis factor (TNF), and the cryptidins.
- Absorptive enterocytes
- Enteroendocrine cells produce the gastrointestinal hormones .

AETIOLOGY

Ileal perforation is a serious and common complication of many systemic illness.

According to literature , in developed countries the common causes are

- Foreign bodies
- Crohns
- Drugs
- Radiotherapy
- Congenital malformations
- Malignancies
- Diverticula
- Ischemic enteritis
- Non specific

Karmakar et al[(1)], the causes of ileal perforation were as follows (1)

| | |
|---|---|
| **Typhoid** | **17** |
| **Non specific** | **7** |
| **Round worm** | **2** |
| **Meckels** | **1** |
| **TB** | **1** |
| **Trauma** | **2** |
| **Total** | **30** |

**The results of the karmakar et al study can be summarised as follows :**

Most common cause -- Typhoid followed by perforation due to non specific. Nil case reported due to malignancy

**Indian study by Bhalerao[(11)] et al**.

| | |
|---|---|
| Nonspecific | 18 |
| Typhoid | 8 |
| TB | 3 |
| Trauma | 3 |
| Diverticulitis | 2 |

In the study by Bhalero ---- Non specific > Typhoid

**Series by Wani et al[(41)].**

| | |
|---|---|
| Typhoid | 49 |
| Non specific | 21 |
| Obstruction | 5 |
| TB | 3 |
| Radiation enteritis | 1 |

Here Typhoid > Non specific perforation

Hence overall the common cause of ileal perforation in tropics ----

TYPHOID FEVER

**TYPHOID PERFORATION :-**

**INCIDENCE**

The documented rate of enteric perforation ranges from 0.5% to 78.6% [14,16,17,18,19,20].

**The following were the incidences obtained in various studies**

| AUTHOR | YEAR | COUNTRY | % |
|---|---|---|---|
| Purohit | 1976 | India | 0.5 |
| Thakkar | 1979 | India | 3.77 |
| Arigbabu | 1980 | Nigeria | 78.6 |
| Hadley | 1984 | SouthAfrica | 4 |
| Santillana | 1991 | Peru | 7.8 |

**AGE AND SEX :-**

Typhoid perforation has a preponderance towards male sex & younger age groups especially in the second and third decades of life [4,5,26].

| AUTHOR | YEAR | M:F | AGE in yrs | MEAN AGE |
|---|---|---|---|---|
| Thakkar | 1979 | 5.2:1 | 6-54 | - |
| Eggelston | 1979 | 3.1:1 | 4-65 | 28 |
| Tarpley | 1989 | 2.3:1 | - | 27.1 |
| Santillana | 1991 | 6:1 | 4-56 | - |
| Mock | 1993 | 2.4:1 | 2-72 | 16.7 |
| Tacyildiz | 1996 | 2.4:1 | 3-70 | 27 |
| Singh | 1996 | 3:1 | - | 14.2 |

**PATHOLOGY :-**

The causative organism of typhoid fever is SALMONELLA TYPHI , a gram negative bacillus. The bacillus passes through the peyers patches and multiplies in the reticuloendothelial system for about 10-14 days. The onset of clinical symptoms correlate with the seeding of the organism into the blood stream.After 14 days ie 2 weeks the bacillus reaches the gut via the blood stream and localises in the peyers patches. Ulceration and necrotic mesenteric lymphadenitis occurs which in course of time leading on to perforation of peyer's patches[17,27,28] commonly encountered in the 2 nd week of the onset of illness [17,24,25].

LIZZARALDE et al [25] studied the timing of perforation in a series of 59 children which is as follows ;

| TIMING in wks | No. | % |
|---|---|---|
| First | 8 | 13.5 |
| Second | 32 | 54.2 |
| Third | 13 | 22 |
| Fourth | 6 | 10 |

**MACROSCOPY :-**

The most consistent feature is a mesenteric lymphadenopathy. Longitudinal ulcers which are characteristic of enteric perforation are seen in the terminal part of the ileum and caecum. The diameter of the perforations varies between 5-8 mm. Hadley in his study found a large number of perforations less than 5mm [17].

**MICROSCOPY :**

There will be both local and systemic marked proliferation of the lymphoid follicles of reticuloendothelial cells. There also occurs accumulation of mononuclear cells and these macrophages are found as aggregates of small nodules filled in the center with red blood cells which is commonly referred to as Erythrophagocytosis. Rarely typhoid bacilli may also be seen within the aggregates[29].

**CLINICAL FEATURES :-**

The symptoms of typhoid fever include fever , G.I. symptoms , respiratory symptoms ,headache . Signs like rose spots , splenomegaly , and relative bradycardia. If untreated or there is a delay in diagnosis the patients can enter into the stage of complications like intestinal perforation , pneumonia, cholecystitis, myocarditis, bleeding [28,30]. Vomiting , sudden worsening of abdominal pain , and distension warns the onset of

perforation.These signs and symptoms will be masked in a toxic patient making a delay in diagnosis [18].

**DIAGNOSIS :-**

In endemic areas diagnosis of typhoid fever is mainly centered around clinical suspicion [17,18,19,31]. The diagnosis of complication like intestinal perforation can be made out by the presence of free gas under the diaphragm, pneumoperitoneum , pus in abdominal paracentesis and peritoneal lavage to detect pus or bile [11].

The laboratory diagnosis of typhoid fever is usually by blood culture in the first week , serology by widal test in the second week , stool & urine culture in the third and fourth week of illness respectively.

**SEROLOGY :-**

Widal test being the most commonly performed test which tend to measure the capsular and flagellar antibodies against the antigens of S.typhi & S.paratyphi. the test is positive between 7 – 10 days of onset of illness. In endemic regions this test is of limited value. Rising titres are considered significant[33] esp. four fold rise in titres[38] have a higher positive predictive value of diagnosing this fever. Patients with history of typhoid fever could have a transient rise in titres which is unrelated to fever is mainly due to the anamnestic response.

**CULTURE OF ORGANISM :-**

The specimens like blood , urine, stool , and bone marrow aspirate can be used to isolate the organism.In the absence of illness blood culture positivity can be seen in about 80% during the first week of illness which decreases to about 25% in the consequent weeks

By the third week of illness about in 75% of the patients stool cultures show a positive result which were previously negative in the first week. Duodenal aspirates, biopsy from rose spots , sputum ,pus in suppurative lesions, CSF constitute the various other sources of culture[35].

**HISTOPATHOLOGY :**

The presence of mononuclear macrophage cells and RBC s within the phagocytes (Erythrophagocytosis) confirms the diagnosis of enteric fever[29].

**NEWER DIAGNOSTIC MODES :-**

Nowadays several newer diagnostic modes have reached the labs. Some of them are Indirect fluorescent Vi antibody, Indirect hemagglutination ,ELISA which have higher sensitivity and specificity than widal test[33] .Much more rapid detection can now be obtained with the monoclonal antibody use against Salmonella typhi flagellin .For detection of salmonella in blood DNA probes for specific antigens are also available

**TREATMENT :-**

Appropriate treatment of typhoid perforation was under controversy till 1957. Many surgeons advocated surgical treatment but with the advent of chloramphenicol into the market this trend has changed towards the conservative line of management.

In 1959 Huckstep insisted on conservative management on the basis of Oschner – Scherren regimen and pointed out the reasons which were as follows ;

Cons :- The friability of the terminal ileum and the liability for multiple perforations makes it bad for holding the sutures.

Pros :- The sterilisation of the bowel contents by the chloramphenicol therapy and the localisation of the perforation by the adjacent bowel loops itself[36].

Absence of localisation in patients have led Hook et al [37] to recommend surgical treatment in those patients.Nowadays surgical treatment [38 ,39] is the best option available as conservative management is associated with significant mortality.

## SURGICAL TREATMENT :-

Preoperative resuscitation of patients by improving the hydration status with intravenous fluids , appropriate antibiotics , and total parenteral nutrition has considerably reduced the mortality rate from 28% to 10% in one study conducted by Tacyildiz et al [(20)].

The various surgical options available are ;

- Drainage of the peritoneal cavity
- Simple closure
- Wedge resection and closure
- Resection anastomosis
- Ileotransverse colostomy
- Ileostomy

## DRAINAGE OF PERITONEAL CAVITY :-

It is mainly indicated during the period of resuscitation and preparation before surgery in moribund patients [(40)]. Here the flank drains are inserted under local anesthesia. As a sole procedure it has an unacceptably higher mortality rate.So it is mainly used as a bridging measure or as a preliminary measure before surgery in moribund patients**.**

**SIMPLE CLOSURE :-**

Archampong[31] recommended simple freshening of edges and primary closure . A significant rate of mortality of about 17.3% was associated with this procedure.The recommendations by Talwar et al [41] is primary simple closure and limited surgery.

**WEDGE RESECTION AND CLOSURE :-**

Here a wedge resection of ileal tissue is done around the perforation and the ileal defect is closed in two layers transversely . A mean mortality rates of about 4% has been reported with this procedure . Reports by Ameh et al [42] advocated an association of very high mortality rate with this procedure.

**RESECTION ANASTOMOSIS :-**

Here resection of the affected segment and primary anastomosis has been advocated by some authors .Studies by Ameh et al recommends primary resection anastomosis over wedge resection & simple closure [42] . Jarret & Gibbney recommends resection only for multiple ileal perforations[28,40].

**ILEOTRANSVERSE COLOSTOMY :-**

The above procedures can be combined with a side to side ileo transverse colostomy.Eggleston and Lizzaralde recommends terminal ileal

closure and end to side ileo transverse colostomy which decreases the incidence of complications [43].

This anastomosis helps by diverting the faecal matter from reaching the diseased ileum and this reduces the risk of complications [25]

**TUBE ILEOSTOMY ;-**

It was first introduced in 1940 by Lozoya . It was carried by passing a 24F foley catheter through the least edematous parts of the ileum. It is versatile ,simple , quick in decompressing the bowel and also prevents further decontamination of peritoneum by the faecal matter .Maloney in Vietnam , Kaul, Ardhanari , Rangabashyam also reported a reduced rate of complications with this procedure.Bhalero et al and Santallina recommends exterioration of suture line preventing further bowel contamination in case of reperforation [10,18].If there is a confirmed preoperative diagnosis of perforation, Talwar et al recommends a Rutherford Morrison incision which is associated with a reduced rate of complications [45].Here the laprostomy comes into play in case of fulminant abdominal sepsis due to faecal fistula formation. It helps in draining the pus out of peritoneal cavity thus preventing further rise in abdominal pressure . The disadvantage of this procedure is the perforation of the exposed bowel and incisional hernia formation[45].

**MEDICAL THERAPY :-**

The gold standard drug for the treatment of typhoid fever was Chloramphenicol used in the dose of 3-4 g /day or about 50 mg/kg for paediatrics which may be reduced to 2g /day or 30mg / kg in children once the fever subsides. Rapid response to the drug occurs in 24-48 hrs and treatment duration shouldn't be less than 2 weeks.Chloramphenicol resitance has emerged as a challenge and so Quinolones has replaced chloramphenicol as drug of choice[47,48]**.** Ciprofloxacin in a dose of 250 – 750 mg BD is used . The alternative drug used is Ceftriaxone[47]. Amoxicillin and Co trimoxazole has also been tried[46] .In patients with perforation peritonitis , Metronidazole and gentamicin should also be added to typhoid drugs[47].Vaidyanathan et al found that metronidazole greatly reduced mortality when added to anti-typhoid drugs [49].

**COMPLICATIONS :-**

Typhoid perforation has a very high complication rates of 28% - 80% [17,18,24,26].

Keenan et al reports in 1981

| COMPLICATIONS | % |
|---|---|
| Chest infection | 55 |
| Sepsis | 46 |
| Wound infections | 33 |
| Recurrent typhoid | 11 |
| Reperforation | 5 |
| G.I. fistula | 5 |
| Wound dehiscence | 5 |

Santillana et al reports in 1991

| | |
|---|---|
| Wound infections | 40.6 |
| Respiratory | 10.4 |
| Renal failure | 2.3 |
| G.I. fistula | 2.1 |
| Melena | 2 |
| Icterus | 2.1 |
| Sepsis | 3.1 |
| Parotid abscess | 0.7 |

**MORTALITY :-**

A significantly high mortality rates have been found in association with typhoid perforation and this is elevated much more in patients managed conservatively**.**

**PROGNOSIS :-**

Factors known to affect survival include[28]

- Female sex
- Multiple perforations
- Late presentations
- Age over 40 yrs

Archampong et al[19] reported following factors to affect survival :-

- Duration of illness
- Urine output before surgery
- Serum potassium
- Perforation – operation interval
- Blood urea

Talwar et al in his study found out that the following factors increases the mortality :-

- Perforation – surgery time interval
- Faeculent peritonitis

Ameh et al reported the relationship between the type of procedure and mortality **:-**

| **PROCEDURES** | **MORTALITY (% )** |
|---|---|
| Simple closure | 50 |
| Wedge resection | 62 |
| Resection anastomosis | 36 |

# TRAUMATIC PERFORATION

## HISTORICAL BACK GROUND

Aristotle was the first to describe visceral injury from non penetrating injury by noting that the intestine of the deer was so delicate that it might be ruptured by a slight external blow without injuring the skin.

In 1836 Baudens published the results of his experiences in the French-Algerian war and suggested “Bold operations” in some cases of gun shot wounds of the abdomen.He was probably the first to have performed Laprotomy for gunshot wounds of the abdomen having operated on two patients in 1830,one of whom survived.

Sims was the first in the United States to advocate surgical intervention for penetrating organ injury.Walter,in 1859,and Kinlock in 1863,were the first to perform abdominal operations for nonpenetrating and penetrating injuries respectively.

## ETIOLOGICAL CLASSIFICATION OF ABDOMINAL INJURIES

Abdominal injuries can be classified as

(i) PENETRATING

(ii) NON-PENETRATING

## PENETRATING

a. Stab wounds

b. Gun shot wounds (Velocity)

c. Shot gun wounds(Range)

d. Other(Sharpnel,Picket,Stake,Glass)

## NON PENETRATING

a. Blunt injury

b. Crush injury

c. Blast injury

d. Seatbelt injury

Knives,Screw drivers, scissors,Pencil,Glass bottles are commonly used to inflict stab wounds in our country.Contrary to the west countries gun shot wounds are less common than stab wounds.

Today the incidence of non penetrating abdominal trauma,is increasing primarily because of the soaring automobile accidents.Automobile is responsible for atleast 50% of non penetrating abdominal injuries.

According to DiVincenti and associates report:

Road Traffic Accidents - 74%

Blow to the abdomen - 14%

Falls - 9%

Other causes - 3%

Non penetrating abdominal trauma is associated with 20 to 30 percent mortality rate, much of which is attributed to associated injuries of head and chest and fractures of the extremities.

## MECHANISM OF INJURY

### PENETRATING INJURIES

Gun Shot Wound –Civilian gun shot wounds are usually caused by low velocity pistols,whereas,military bullet wounds are of the high velocity type and result in extreme tissue destruction that requires wide and thorough debridement.

Physical factors involve the Kinetic energy imparted to the body by the missile.

Kinetic energy of a missile is expressed by the formula

E = 0.5 x mv2 x g x 7000

m = mass of missile

v = Velocity of missile

g = gravitational acceleration

The amount of energy imparted to the body is the difference between the kinetic energy of the missile entering minus that when leaving thebody. This energy is dissipated by the movement of the tissues in a perpendicular direction from the trajectory of the bullet.

Characteristics of the tissue determine the extent of destruction.Fascia,skin and Lung reveal little devitalisation when struck by high velocity missile,whereas solid tissues such as muscle,bone, liver and spleen are violently disorganized and devitalized.

The common low velocity injury is stabbing.In this injury the kinetic energy is low. Thus deep penetration of the abdominal cavity is statistically the exception rather than the rule.

The shape,size and length of the instruments are important in estimating the amount of the damage that might have caused.

## NON-PENETRATING INJURIES

Mechanism of nonpenetrating visceral injuries include crushing, shearing and bursting forces.

First is the crushing of the organ against the posterior abdominal wall,especially the anterior ridge in the midline produced by the vertebral bodies.

A sharp shearing force may suddenly be applied to both solid and hollow organs resulting in tears with perforation or haemorrhage or both.

Thirdly an intra-abdominal hollow viscus can be burst open by asudden increase in its intra-luminal pressure.

A sudden application of pressure is more apt to rupture solid than hollow viscera ,thus accounting for the greater incidence of solid organ injury.

The more elastic tissue of the young tolerate, trauma better than the resilient tissues of the aged.A strong firmly muscled abdominal wall constitutes a better barrier than the flaccid ,relaxed abdomen of the old or intoxicated.

**Seatbelt injuries**

Since Kulowski and Rost in 1956 first attributed a case of intestinal obstruction to a previously ocurred seatbelt injury.Numerous cases of the seatbelt Syndrome have appeared in the literature.

The method of injury to the Bowel in this syndrome involves direct trauma that results in seromuscular tears and closed-loop obstructions that temporarily increases intra luminal pressure,resulting in intestinal rupture .Shearing and torsion forces are probably also active.Besides the intestines and mesentry,practically all abdominal structures,including the gravid uterus,have been injuried by seat belts.The terminal ileum is the most common location for the intestinal injuries.

**SMALL BOWEL INJURY SCALE: (BY OIS COMMITTEE OF AAST, 1990)**

| Grade | Type of injury | Description of injury |
|---|---|---|
| I | Haematoma | Contusion or Haematoma without devascularization. |
| | Laceration | Partial thickness, No perforation |
| II | Laceration | Laceration < 50% of circumference |
| III | Laceration | Laceration> 50% of circumference without transection |
| IV | Laceration | Transection of small Bowel |
| V | Laceration | Transection of small Bowel, with segmental tissue loss |
| VI | Vascular | Devascularized segment |

Incidence of ileal perforations in a series of traumatic perforations stabs or gunshot wounds.

| AUTHORS | No. of traumatic perforations | No. of ileal perforations |
|---|---|---|
| Kaul et al[52] | 24 | 10 |
| Karmakar[1] | 30 | 13 |
| Scully et al[53] | 20 | 2 |
| Paran[55] | - | 2 |

**DIAGNOSIS :-**

Based on

- Clinical examination
- X- ray
- Four quadrant needle aspiration

X-ray chest or abdomen can reveal free air under the diaphragm.

Abdominal paracentesis was positive in 85 % of patients with small bowel perforation in a study by Koul et al[52].

**General guides for treatment**

1. Short single tear (not more than 4 cm) are repaired in the transverse axis of the gut.
2. Longer single tear may be repaired in the long axis.
3. Contusions are better left alone

**Resection is called for**

1. When associated mesenteric lesion has devitalized the damaged section
2. When the injury has mangled the intestine
3. When several perforations are grouped close together

**TUBERCULOSIS :-**

Tuberculosis is usually an under diagnosed cause for ileal perforation.

| AUTHORS | TOTAL NO. OF ILEAL SPECIMENS | NO. OF SPECIMENS POSITIVE FOR TB |
|---|---|---|
| Gera et al[57] | 113 | 9 |
| Karmakar et al[1] | 30 | 1 |

**PATHOGENESIS :-**

In the past it was thought to be caused by the ingestion of Mycobacterium bovis infected milk.

Bowel is being affected by ingestion of infected sputum or saliva in patients following pulmonary tuberculosis. But nowadays the incidence of intestinal tuberculosis without the evidence of significant pulmonary lesion is on the increasing trend owing to the increase in the incidence of HIV infection.

Free intestinal perforation accounts for about 7 % of abdominal TB patients and associated with a high mortality rate of about 30% and a poor prognosis .

**Two types of lesion**

- Ulcerative
- Hypertrophic

In hypertrophic variety the segment of ileum proximal to strictures is the one most commonly to perforate[58] .

Wig et al[59] reported 10 cases of perforation through the ulcerative variety.

Incidence of perforation varies between 1- 10% .

Kakar et al found multiple perforations in 36% of cases and strictures in 72.6%[58] .

**DIAGNOSIS :-**

The diagnosis is usually made by the visualisation of tubercles in the resected segment of intestine and in mesenteric lymph nodes .Wig et al showed acid-fast bacilli in about 40% of resected specimens. Makanguola et al shown that CT can help in diagnosing intestinal TB in about 80 % of patients.Chest radiography may reveal air under the diaphragm and it may be normal in about 50% of patients with extra pulmonary tuberculosis.

**TREATMENT :-**

Resection-anastomosis is the technique of choice .

Resection of the affected segment with ileo-transverse anastomosis is an alternative procedure .

**MECHANICAL FACTORS :-**

Mechanical etiology of perforation is one in which perforation occurs secondary to a obstruction in the distal part .

**CAUSES :-**

- Obstructing growth
- Hernias
- Intussusception
- Volvulus
- Bands

**PATHOGENESIS :-**

The initial insult is obstruction either by a band or hernia which in course of time leads onto venous outflow obstruction causing odema of the proximal intestinal wall and subsequently gangrene . Increased intraluminal pressure of the proximal gangrenous bowel leads onto rupture and perforation[(27)].

**Chaikof et al[12] in his series of study ,**

| TOTAL NO. OF SMALL BOWEL PERFORATION 76 | NO. OF MECHANICAL CAUSES 18 |
|---|---|
| | Adhesions - 12<br>Hernia - 4<br>Carcinoma - 2 |

**Dixon et al in their reports**

| TOTAL NO OF SMALL BOWEL PERFORATION 54 | NO. OF MECHANICAL CAUSES 13 |
|---|---|
| | Adhesions - 8<br>Malignancy - 2 [2]Volvulus - 1 |

**MALIGNANCY :-**

Small bowel malignancies are very rare causes of all gastrointestinal malignancies accounting for only about 3%. Reported tumors in order of frequency is as follows ;

- Adenocarcinoma
- Carcinoid
- Lymphoma
- Sarcoma

Most common site being Ileum[61].

Most common tumors to perforate being Lymphomas.

| AUTHORS | TOTAL NO OF SMALL BOWEL PERFORATIONS | NO. OF PATIENTS WITH LYMPHOMA[13] |
|---|---|---|
| Rajagopalan & Pickleman | 16 | 2 |

**PATHOGENESIS :-**

Lymphomas has a propensity to grow centrifugally in the bowel wall causing partial or distal obstruction[13] and this being the common site to perforate.

**TREATMENT :-**

Resection of the involved segment and adjoining mesentery[61] .

**INFLAMMATORY BOWEL DISEASE :**

It constitutes crohns disease and ulcerative colitis . In crohn's free wall perforation is a rare complication.Most common site being Ileum. Steinberg et al[62] in their study.

**PATHOGENESIS :-**

During an acute exacerbation in crohns disease ,there occurs a proximal perforation which is usually secondary to a distal obstruction .

**TREATMENT :-**

Simple primary closure is an inappropriate treatment associated with a poor prognosis.

Menguy et al [63] recommends primary excision of the affected segment & formation of a double-barreled ileocolostomy with closure of stoma in later sitting.

**NON SPECIFIC PERFORATION :-**

When the ileal perforation is due to an unknown cause , its called non specific perforation .

| **Authors** | **Total no. of ileal perforation** | **No. of non specific perforation** |
|---|---|---|
| Dixon et al[2] | 54 | 30 |
| Karmakar [1] | 14 | 7 |

Recent studies are giving promising results on the pathogenesis of non specific perforation as sub mucosal vascular emboli[64] and potassium tablets as the factors causing ulceration and subsequent perforation .

**DIVERTICULITIS :-**

Perforation of diverticula per se is one of the rare cause of small bowel perforation.

| **Authors** | **Total no. of ileal perforation** | **No. of perforation due to diverticula** |
|---|---|---|
| Huttunen et al [66] | 24 | 4<br>Ileal diverticulum - 1<br>Diverticulitis - 2<br>Meckel's - 1 |
| Bhalero et al[11] | 32 | 2 |

The incidence of meckel' s diverticulum in general population is about 0.4 – 2.5 %. Ectopic gastric mucosa occurs in about 38 % of meckels diverticula [67] which might ulcerate leading on to perforation [66] .The recommended treatment of choice is resection of the diverticulum along with the adjacent ileum .[12 ,67]

**ISCHEMIC ENTERITIS :-**

Ischemic enteritis is another rare cause of ileal perforation.

| Author | No of ileal perforation | No of ischemic enteritis |
|---|---|---|
| Dixon [2] | 54 | 3 |

Macroscopically the lesion can be divided into 4 stages :-

A. Segmental bluish mucosal discolouration , edema and ulceration

B. Purple circular bands with bowel wall edema

C. Rigid pipe like , lengthier intestinal segment

D. Subsequently the segment appears thin and papery.

Perforation has higher incidence of occurence in the fourth stage. Histologically the specimen shows areas of necrosis, severity of which varies proportionally with stage of the disease [68].

**MISCELLANEOUS :-**

The causes [1,2,3] are

- Radiation enteritis
- AIDS
- Polyarteritis nodosa
- Steroid intake
- Round worm infestations

Remine et al reports as follows :-

- At the occurrence of perforation about 79 patient on Steroids
- A mortality rate of 85.1% was seen in patients taking prednisolone > 20mg /day
- Medical conditions warranting steroids present in 62 % Of patients like Metastatic cancer ,Connective tissue disorders , Myeloproliferative syndrome.
- Perforation risk greatest in the period of first 3 weeks following the initiation of steroid therapy [(69)].

Sunke et al in his case series shown a patient of AIDS with cytomegalo virus as the cause of ileal perforation.

# *METHODOLOGY*

# METHODOLOGY

**Source of Data**:

This study consists of patients admitted from October 2010 to October 2012. 70 patients of ileal perforation who were admitted in Mahatma Gandhi Memorial Government Hospital during this period have been included in the study.

**Inclusion Criteria:**

- All cases of ileal perforation of age > 14 years.
- Traumatic Ileal perforation associated with solid organ injuries
- Ileal perforation associated with Extra-Abdominal injuries

**Exclusion Criteria:**

- Jejunal perforation,
- Gastric & duodenal perforation,
- Appendicular , Caecal , Colonic perforation
- All cases of ileal perforation of age < 14 years
- Ileal perforation treated conservatively
- Gangrenous perforation.

**Study Method**:

Clinical features, investigations, operative procedures done, postoperative morbidity, mortality and its outcome were studied. History of mode of injury, time of injury and special reference to presence of pain, fever, vomiting, abdominal distension, constipation were recorded. Vitals, hydration status, abdominal distension, tenderness, guarding & rigidity, Liver dullness obliteration and presence of free fluid were noted.

Systemic examination of cardiovascular system, respiratory system and central nervous system has been done.

The following investigations had been done as a routine

- Haemoglobin
- TC, DC , ESR
- Bleeding and Clotting time
- Blood sugar ,urea and Serum creatinine & electrolytes
- Chest X-Ray PA view, X-Ray Abdomen Erect view
- Electrocardiogram
- Pus culture in case of wound infection

4 Quadrant Aspiration was done in some cases.

USG Abdomen was taken for whom haemoperitonium was suspected.

CT Abdomen was taken for few cases.

CT Brain was taken for some patients to rule out head injury.

X- Ray of the local region was taken where fracture was suspected.

In all non-traumatic perforations the following additional investigations were done

1. Widal test
2. Blood Culture

When perforation due to non-traumatic causes like TB, Typhoid was suspected lymph node and omentum was taken for biopsy and peritoneal fluid was taken for Culture & Sensitivity and AFB staining. All patients had been resuscitated preoperatively with intravenous fluids and blood transfusion and iv antibiotics. For patients with medical problems Physician opinion was obtained and treated accordingly.

Most cases received Third Generation Cephalosporins like cefotaxime, Ceftriaxone or ciprofloxacin with metronidazole. In the event of gross peritoneal contamination aminoglycosides were added.

Nature of disease , procedure to be done and its prognosis were explained to patient's relatives and written high risk consent was obtained for all cases. All patients underwent laparotomy under Regional or general anaesthesia. Most of the surgeries have been performed by trainee surgeons under supervision of Duty Assistant surgeon. Either Midline or Para median incision was employed. The type of peritoneal contamination, amount of peritoneal fluid aspirated was noted. Thorough laprotomy was done. Number, site and size of perforations and procedure done were noted. The choice of surgical procedure was based on surgeon's preference & unit policy.

The following procedures were done.

1. Simple two layer closure
2. Resection and anastomosis
3. Closure with free or pedicled omental patch

For both 2 layer closure and resection anastamosis , the inner all-coats layer was sutured with polyglactin 910 and outer layer with silk. Thorough peritoneal wash was given routinely, DT was kept in flank region, Post-Operatively , antibiotics were routinely given for 5-8 days unless the diagnosis was typhoid where antibiotics were continued for up to ten days.

Diagnosis of typhoid was made only if Widal test was Strongly positive, or Salmonella typhi was isolated from blood or urine and also if any histopathological evidence of typhoid perforation has been found. When the etiology of perforation was not found, it has been termed as non-specific. Routine dressing done during post operative period. Postoperative complications if any had been noted and treatment given for that. DT removal and suture removal were done depending upon individual cases. The factors influencing morbidity, mortality and outcome were . patients with traumatic causes of perforation associated with any extra-abdominal injuries were treated with appropriative method.

The various parameters had been recorded in a proforma and tabulated. Analysis of result of study was done. Most of the result given in the form of percentage, ‘p’ value significance were used wherever necessary, and conclusions were arrived finally..

# *RESULTS*

# RESULTS

Seventy patients of Ileal Perforation admitted between October 2010 and October 2012 were included in this study. Patients have been categorised into etiological groups namely, Traumatic , Non specific, G.I tuberculosis and typhoid .

**Etiology**

The commonest cause of ileal perforation in my study was Trauma accounting for 87% of the total. 6 patients had non-specific perforations and is the second most common .Two patients were diagnosed to have gastrointestinal tuberculosis with ileal perforation. One patient had history of typhoid fever.

The distribution is shown in Table 1

**Table 1-CAUSES OF ILEAL PERFORATION**

| CAUSE | NO. | PERCENTAGE |
|---|---|---|
| TRAUMATIC | 61 | 87% |
| TUBERCULOSIS | 2 | 3% |
| TYPHOID | 1 | 1% |
| NON SPECIFIC | 6 | 9% |
| TOTAL | 70 | 100% |

Of the traumatic perforation 34 patients were admitted with history of blunt injury and 27 patients admitted for penetrating injury.Blunt injury results from RTA and fall from height. 27.9% accounts for stab injury , 9.8 % accidental injury and 6.6% accounts for Bull Gore injury. The Distribution of Traumatic perforation is shown in table 2:

**Table 2-TRAUMATIC CAUSES OF ILEAL PERFORATION**

| **MODE OF INJURY** | | **NO.** | **PERCENTAGE** |
|---|---|---|---|
| BLUNT INJURY | | **34** | **55.7** |
| PENETRATING INJURY | STAB INJURY | **17** | **27.9** |
| | ACCIDENTAL | **6** | **9.8** |
| | BULL GORE | **4** | **6.6** |
| TOTAL | | **61** | **100.0** |

**Age and Sex Incidence**

The age of patients ranged from 16 to 65. Perforation commonly occurred in the third and fourth decade of life with 58% of patients between the ages of 30 and 50. The male to female ratio was 6.7:1.

Traumatic perforation commonly occurred in the third decades with35.57% of cases in that age group. Male to female ratio was 5.7:1. Non-Specific perforations occurred commonly in the second and fifth decades of life with equal occurrence of 33.3% in each decade and were commoner in

male . The distributions of age and sex of all cases and etiology specific distributions are shown in tables 3 and 4

**Table 3-SEX INCIDENCE IN ILEAL PERFORATION**

| | TRAUMATIC | | NON TRAUMATIC | | TOTAL | |
|---|---|---|---|---|---|---|
| | **NO.** | **%** | **NO.** | **%** | **NO.** | **%** |
| **MALE** | 52 | 85 | 9 | 100 | 61 | 87 |
| **FEMALE** | 9 | 15 | 0 | 0 | 9 | 13 |
| **TOTAL** | 61 | 100 | 9 | 100 | 70 | 100 |

**TABLE 4- AGE INCIDENCE IN ILEAL PERFORATION**

| AGE | ILEAL PERFORATION | | BLUNT INJURY | | PENITRATING INJURY | | TB | | TYPHOID | | NON SPECIFIC | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | **NO.** | **%** | **NO.** | **%** | **NO.** | **%** | **NO.** | **%** | **NO.** | **%** | **NO.** | **%** |
| **<20** | 5 | 7.1 | 4 | 11.7 | 1 | 3.7 | 0 | 0 | 0 | 0 | 0 | 0 |
| **21-30** | 11 | 15.7 | 3 | 8.8 | 6 | 22.2 | 0 | 0 | 0 | 0 | 2 | 33.3 |
| **31-40** | 25 | 35.7 | 10 | 29.4 | 13 | 48.1 | 1 | 50 | 0 | 0 | 1 | 16.6 |
| **41-50** | 16 | 22.8 | 8 | 23.5 | 7 | 25.9 | 0 | 0 | 0 | 0 | 1 | 16.6 |
| **51-60** | 10 | 14.2 | 6 | 17.6 | 0 | 0 | 1 | 50 | 1 | 100 | 2 | 33.3 |
| **>60** | 3 | 4.2 | 3 | 8.8 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| **TOTAL** | **70** | **100** | **34** | **100** | **27** | **100** | **2** | **100** | **1** | **100** | **6** | **100** |

**Symptoms and Signs**

Majority of the patients presented with symptoms and signs of peritonitis. The commonest symptoms encountered were abdominal pain, fever and altered bowel habits. Almost all patient presented with abdominal pain and altered bowel habits was seen in 25.7% of patients.

The commonest signs were guarding and rigidity, obliteration of liver dullness and dehydration. 55% of Patients came with History of penetrating injury presented with soft Abdomen. Typhoid patient gave a history of fever. 31.42 % of patients were in shock . No patients of penetrating injury presented with Shock. 10% of Patients presented with Omenatal Prolapse and 5.71% of patient presented with Bowel Prolapse. Symptoms and signs are shown in tables 5 and 6.

## Table 5-Symptoms of Ileal Perforation

| SYMPTOMS | | ILEAL PERFORATION | | BLUNT INJURY | | PENETRATING INJURY | | TB | | TYPHOID | | NON SPECIFIC | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | NO | % | NO | % | NO. | % | NO | % | NO | % | NO | % |
| ABDOMINAL PAIN | | 70 | 100 | 34 | 100 | 27 | 100 | 2 | 100 | 1 | 100 | 6 | 100 |
| FEVER | | 15 | 21.42 | 9 | 26.47 | 0 | 0 | 2 | 100 | 1 | 100 | 3 | 50 |
| ALT BOWEL HABITS | | 18 | 25.71 | 9 | 26.47 | 0 | 0 | 2 | 100 | 1 | 100 | 6 | 100 |
| VISERA EVISCERATED | OME | 7 | 10 | 0 | 0 | 7 | 25.9 | 0 | 0 | 0 | 0 | 0 | 0 |
| | BOW | 4 | 5.71 | 0 | 0 | 4 | 14.8 | 0 | 0 | 0 | 0 | 0 | 0 |

## Table 6-SIGNS IN ILEAL PERFORATION

| SIGNS | ILEAL PERFORATION (70) | | BLUNT INJURY (34) | | PENITRATING INJURY (27) | | TYPHOID (1) | | TB (2) | | NON SPECIFIC (6) | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | NO | % | NO | % | NO. | % | NO | % | NO | % | NO | % |
| SOFT | 16 | 22.8 | 0 | 0 | 15 | 55.5 | 0 | 0 | 0 | 0 | 1 | 16.7 |
| GUARDING & RIGIDITY | 54 | 77.14 | 34 | 100 | 12 | 44.4 | 1 | 100 | 2 | 100 | 5 | 83.3 |
| LIVER DULLNESS OBLITERATION | 53 | 75.7 | 32 | 94.11 | 13 | 48.1 | 1 | 100 | 2 | 100 | 5 | 83.3 |
| SHOCK | 22 | 31.42 | 19 | 55.88 | 0 | 0 | 1 | 100 | 1 | 50 | 1 | 16.7 |

## Investigations

X-Ray: Pneumoperitoneum in chest and erect abdominal x-rays was seen in 75.7% of patients. Features of intestinal obstruction, including dilated bowel loops with air-fluid levels in erect abdominal x-ray were also seen in a significant percentage of patients.Ultrasound abdomen taken for some blunt injury abdomen patients which revealed Haemoperitoneum and solid organ injury. 4 Quadrant aspiration done for some patients.Biopsy was taken from Omentum and nodes of two patients who showed features of Tuberculosis.

**Table 7-AIR UNDER DIAPHRAGM**

| AIR UNDER DIAPHRAGM | ILEAL PERFORATION (70) | | BLUNT INJURY (34) | | PENITRATING INJURY (27) | | TYPHOID (1) | | TB (2) | | NON SPECIFIC (6) | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | NO. | % | NO. | % | NO. | % | NO. | % | NO. | % | NO. | % |
| YES | 53 | 75.7 | 32 | 94 | 13 | 48 | 1 | 100 | 2 | 100 | 5 | 83 |
| NO | 17 | 24.3 | 2 | 6 | 14 | 52 | 0 | 0 | 0 | 0 | 1 | 17 |

## Lag Period

The time between the onset of trauma and the surgical intervention was within 24 hrs in about 67.14% of patients. There was no significant difference in the mean lag periods of patients of typhoid or non-specific perforations. Traumatic perforations were found to have a significantly reduced lag period because of early diagnosis.Pattern of lag period is shown in table 8.

**Table 8-LAG PERIOD IN ILEAL PERFORATION**

| LAG PERIOD | ILEAL PERFORATION | | BLUNT INJURY | | PENITRATING INJURY | | TB | | TYPHOID | | NON SPECIFIC | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | NO. | % | NO. | % | NO. | % | NO. | % | NO. | % | NO. | % |
| **<24 HRS** | 47 | 67.14 | 23 | 67.64 | 24 | 88.8 | 0 | 0 | 0 | 0 | 0 | 0 |
| **24-48 HRS** | 6 | 8.5 | 2 | 5.9 | 3 | 11.2 | 0 | 0 | 0 | 0 | 3 | 50 |
| **>48 HRS** | 17 | 24.28 | 9 | 27.64 | 0 | 0 | 2 | 100 | 1 | 100 | 3 | 50 |

## Surgical Procedures

Simple 2-layer closure was the commonest procedure done in about 70% of patients. Closure with omental patch was done in 10% and resection anastomosis in 18.57% of patients. Different types of surgical procedure done in ileal perforation is shown in table 9.

### Table 9- SURGICAL PROCEDURES DONE IN ILEAL PERFORATION

| PROCEDURE | ILEAL PERFORATION (70) | | BLUNT INJURY (34) | | PENITRATING INJURY (27) | | TYPHOID (1) | | TB (2) | | NON SPECIFIC (6) | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | NO | % | NO. | % | NO. | % | NO | % | NO | % | NO | % |
| TWO LAYER CLOSURE | 49 | 70 | 22 | 64.7 | 25 | 92.6 | 1 | 100 | 0 | 0 | 1 | 16.67 |
| RESECTION & ANASTOMOSIS | 13 | 18.57 | 7 | 20.5 | 2 | 7.4 | 0 | 0 | 0 | 0 | 4 | 66.66 |
| CLOSURE WITH OMENTAL PATCH | 7 | 10 | 5 | 14.5 | 0 | 0 | 0 | 0 | 1 | 50 | 1 | 16.67 |
| TUBE DRAINAGE | 1 | 1.43 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 50 | 0 | 0 |

**Number and Site of Perforation**

**About 70% of patients presented with a single perforation .**

Multiple perforations occurred only in 8.6% of patients.

Over 72% of perforations were within 2 feet (60 cms) from the ileocaecal junction and 57.14% within 30 cms. Most of the patients had perforation of size < 1cm . Number and site of perforation is shown in table 10 and 11.

**TABLE 10-NUMBER OF PERFORATIONS**

| NUMBER OF PERFORATION | ILEAL PERFORATION (70) | | BLUNT INJURY (34) | | PENITRATING INJURY (27) | | TYPHOID (1) | | TB (2) | | NON SPECIFIC (6) | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | NO. | % | NO. | % | NO. | % | NO | % | NO | % | NO | % |
| ONE | 49 | 70 | 27 | 79.4 | 20 | 74 | 0 | 0 | 0 | 0 | 2 | 33.33 |
| TWO | 11 | 15.7 | 1 | 2.9 | 5 | 18.5 | 1 | 100 | 1 | 50 | 3 | 50 |
| THREE | 4 | 5.7 | 1 | 2.9 | 1 | 3.75 | 0 | 0 | 1 | 50 | 1 | 16.67 |
| FOUR | 6 | 8.6 | 5 | 14.7 | 1 | 3.75 | 0 | 0 | 0 | 0 | 0 | 0 |

**TABLE 11-DISTANCE OF PERFORATION SITE FROM ILEO CAECAL JUNCTION**

| DISTANCE | ILEAL PERFORATION (70) | | BLUNT INJURY (34) | | PENITRATING INJURY (27) | | TYPHOID (1) | | TB (2) | | NON SPECIFIC (6) | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | NO. | % | NO. | % | NO. | % | NO. | % | NO | % | NO | % |
| <30 CM | 40 | 57.14 | 29 | 85.2 | 3 | 11.12 | 1 | 100 | 2 | 100 | 5 | 83.3 |
| 30-60 CM | 10 | 14.2 | 5 | 14.8 | 4 | 14.8 | 0 | 0 | 0 | 0 | 1 | 16.7 |
| >60 CM | 20 | 28.5 | 0 | 0 | 20 | 74 | 0 | 0 | 0 | 0 | 0 | 0 |

## Complications

Complications occurred in majority of cases . The common complications noted were wound infection, wound dehiscence, pelvic abscess and respiratory complications.The commonest complication encountered was wound infection which is seen in about 61.4% of patients with no significant difference with regards to their etiology .The highest complication rate of wound infection of about 92.3% was seen with resection anastomosis and in simple closure it was about 47%. Complication in relation to causes and modes of surgery are shown in table 12 and 13.

**TABLE 12-COMPLICATION IN RELATION TO CAUSES**

| COMPLICATIONS | ILEAL PERFORATION (70) | | BLUNT INJURY (34) | | PENITRATING INJURY (27) | | TYPHOID (1) | | TB (2) | | NON SPECIFIC (6) | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | NO. | % | NO. | % | NO. | % | NO. | % | NO. | % | NO. | % |
| WOUND INFECTION | 43 | 61.4 | 25 | 73.5 | 9 | 33.3 | 1 | 100 | 2 | 100 | 6 | 100 |
| WOUND GAP | 9 | 12.8 | 7 | 20.5 | 1 | 3.7 | 0 | 0 | 0 | 0 | 1 | 16.66 |
| PELVIC ABSCESS | 18 | 25.7 | 12 | 35.2 | 1 | 3.7 | 0 | 0 | 1 | 50 | 4 | 66.66 |
| RESPIRATORY COM | 18 | 25.7 | 12 | 35.2 | 3 | 11.1 | 0 | 0 | 1 | 50 | 2 | 33.33 |
| LEAK | 3 | 4.2 | 1 | 2.9 | 0 | 0 | 1 | 100 | 0 | 0 | 1 | 16.66 |
| DEATH | 4 | 5.7 | 1 | 2.9 | 0 | 0 | 1 | 100 | 1 | 50 | 1 | 16.66 |

**TABLE 13-COMPLICATION IN RELATION TO SURGICAL PROCEDURE**

| COMPLICATIONS | TWO LAYER CLOSURE (49) | | RESECTION & ANASTOMOSIS (13) | | CLOSURE WITH OMENTAL PATCH (7) | | TUBE DRAINAGE (1) | | TOTAL (70) | |
|---|---|---|---|---|---|---|---|---|---|---|
| | NO. | % | NO. | % | NO. | % | NO. | % | NO | % |
| WOUND INFECTION | 23 | 47 | 12 | 92.3 | 7 | 100 | 1 | 100 | 43 | 61.4 |
| WOUND GAP | 3 | 6.1 | 5 | 38.4 | 1 | 14.2 | 0 | 0 | 9 | 12.9 |
| PELVIC ABSCESS | 6 | 12 | 6 | 46.1 | 5 | 71.4 | 1 | 100 | 18 | 25.7 |
| RESPIRATORY COMPLICATON | 10 | 20 | 4 | 30.7 | 3 | 42.8 | 1 | 100 | 18 | 25.7 |
| LEAK | 2 | 4 | 1 | 7.6 | 0 | 0 | 0 | 0 | 3 | 4.2 |
| DEATH | 2 | 4 | 1 | 7.6 | 0 | 0 | 1 | 100 | 4 | 5.7 |

## Hospital Stay

Resection and anastamosis took a longer time than the other procedures but the difference was not statistically significant.

Median hospital stay was 18.2 days in patients undergone two layer closure and 20.1 days in resection anastomosis .There was no significant difference in the hospital stay of patients undergoing different surgical procedures.

**TABLE 14-DURATION OF HOSPITAL STAY**

| PROCEDURE DONE | MEAN DURATION IN DAYS |
|---|---|
| TWO LAYER CLOSURE | 18.2 |
| CLOSURE WITH OMENTAL PATCH | 19.8 |
| RESECTION & ANASTOMOSIS | 20.1 |

**Mortality and Morbidity** :-

The total mortality rate was 14%. Mortality rates in patients of blunt injury and non-specific perforations were 2.9% and 16% respectively. No patients of penetrating traumatic perforation died and 1 patient of tubercular perforation expired. The differences in mortality were not found to be statistically significant. Septicemia, respiratory Complications were the commonest causes of death. Morbidity and Mortality in relation to disease and surgical procedure are shown in Table 15 and 16.

## TABLE 15-MORBIDITY AND MORTALITY IN RELATION TO DISEASE

| | ILEAL PERFORATION (70) | | BLUNT INJURY (34) | | PENITRATING INJURY (27) | | TYPHOID (1) | | TB (2) | | NON SPECIFIC (6) | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | NO. | % | NO. | % | NO. | % | NO. | % | NO. | % | NO. | % |
| MORTALITY | 4 | 5.7 | 1 | 2.9 | 0 | 0 | 1 | 100 | 1 | 50 | 1 | 16.6 |
| MORBIDITY | 47 | 67 | 25 | 73.5 | 13 | 48.1 | 1 | 100 | 2 | 100 | 6 | 100 |

## TABLE 16-MORBIDITY & MORTALITY IN RELATION TO SURGICAL PROCEDURE

| | TWO LAYER CLOSURE (49) | | RESECTION & ANASTOMOSIS (13) | | CLOSURE WITH OMENTAL PATCH (7) | | TUBE DRAINAGE (1) | | TOTAL (70) | |
|---|---|---|---|---|---|---|---|---|---|---|
| | NO. | % | NO. | % | NO. | % | NO. | % | NO. | % |
| MORTALITY | 2 | 4.08 | 1 | 7.7 | 0 | 0 | 1 | 100 | 4 | 5.7 |
| MORBITY | 30 | 61.22 | 11 | 84.6 | 7 | 100 | 1 | 100 | 49 | 70 |

## LAG PERIOD AND ITS COMPLICATIONS

Most patients presented with peritonitis >24 hrs were associated with increased morbidity and mortality. 88.23% of patients having lag period of >48hrs developed complications.

## TABLE 17-RELATION OF LAG PERIOD WITH COMPLICATIONS

| LAG PERIOD | TOTAL (70) | NO. OF PATIENTS HAVING COMPLICATIONS (47) | | DEATH (4) | |
|---|---|---|---|---|---|
| | NO. | NO. | % | NO. | % |
| **<24 HRS** | 47 | 28 | 59.5 | 0 | 0 |
| **24-48 HRS** | 6 | 4 | 66.66 | 0 | 0 |
| **>48 HRS** | 17 | 15 | 88.23 | 4 | 23.5 |

## Prognostic factors

The type of surgical procedure did not affect the mortality and morbidity in ileal perforations and also in etiology specific analysis. Risk factors influencing the mortality were found to be age >50 yrs , faecal peritonitis , and a lag period of >48 hrs and all the mentioned factors had a statistically significant p value .

Morbidity rates were found to be influenced by factors such as age > 50 yrs , preoperative shock , faecal peritonitis and lag period of >48 hrs .Of which, Faecal periotinitis shown very significant “p” value <0.001

In patients of non-specific perforations lag period showed only a trend towards significance but was not that much statistically significant. Prognostic factors affecting morbidity and mortality are shown in Table 18 and 19.

**TABLE 18-RISK FACTOR FOR MORBIDITY**

| **FACTORS** | **MORBIDITY (47)** | **NO MORBIDITY (23)** | **P VALUE** |
|---|---|---|---|
| AGE > 50 | 9 | 0 | **0.05** |
| MALES | 40 | 21 | 0.7 |
| FEMALES | 7 | 2 | |
| SHOCK | 19 | 0 | **< 0.05** |
| MULTIPLE PERFORATIONS | 12 | 9 | > 0.05 |
| FAECAL PERITONITIS | 9 | 0 | **< 0.05** |
| LAG PERIOD > 48 HRS | 15 | 2 | **< 0.05** |

## TABLE 19-RISK FACTORS FOR MORTALITY

| FACTORS | MORTALITY (4) | NO MORTALITY (66) | P VALUE | |
|---|---|---|---|---|
| AGE > 50 | 3 | 6 | **0.02** | **<0.05** |
| MALES | 4 | 57 | 1.00 | >0.05 |
| FEMALES | 0 | 9 | 1.00 | >0.05 |
| SHOCK | 4 | 15 | **0.06** | **>0.05** |
| MULTIPLE PERFORATIONS | 4 | 17 | 0.0793 | >0.05 |
| FAECAL PERITONITIS | 4 | 5 | **0.0004** | **<0.001** |
| LAG PERIOD > 48 HRS | 4 | 13 | **0.04** | **<0.05** |

# *DISCUSSION*

# DISCUSSION

The commonest cause of ileal perforation in this series was traumatic perforation accounting for about 87% of cases categorised separately as blunt injury contributing to about 55.7% of total traumatic perforation . 8.25% of ileal perforations reported by Karmakar were due to trauma [1]. The increasing rates of road traffic accidents and civil violence have contributed to this rising incidence of traumatic perforations.

When the etiology of the perforation was unidentifiable it was termed as non-specific perforation. Non-specific perforation was the second commonest cause in this study accounting for 9% of cases. Three patients of non-specific perforation had fever before the onset of abdominal symptoms. Widal test, blood cultures and Histopathological reports were not suggestive of typhoid. These cases might be the undiagnosed cases of typhoid. Non-specific perforations were the commonest cause of ileal perforation in the case series by Dixon and Bhalerao [2,11].

Typhoid fever was the commonest cause of ileal perforation in tropical countries. Typhoid fever accounted for 56.6% of cases of ileal perforation in the case series by Karmakar et al [1].

Malignancy and mechanical causes are the commonest causes of small bowel perforations in the western world. In case series by Dixon et al lymphomas and mechanical causes accounted for 40.7% of perforations [2]. In the series by Orringer [3] malignancy was the commonest cause . There were no cases of typhoid perforations in both of the series [2,3].

There was a male preponderance in this study with the male: female ratio being 6.7:1. This preponderance was seen irrespective of the aetiological factors.

Earlier literatures published also shows a similar picture with reported ratios from 2.3:1 to 6.1:1[5,16,18,21,22,23,24,25] .

In the case series by Eggleston et al typhoid perforations occurred in the second and third decade of life [26]. In this study about 58% of cases were in a similar age group occurring between the third and fourth decade of life.

Majority of the patients presented with features suggestive of peritonitis. Patients of both traumatic and non-specific perforations had similar clinical presentation with regards to abdominal symptoms and signs. Patients with traumatic perforation had abdominal pain , altered bowel habits and fever . Examination revealed guarding and rigidity, obliteration of liver dullness and shock . 22 patients were in shock at the time of admission.

Eggleston reported that most patients of typhoid perforation had symptoms of fever, malaise and sudden increase in abdominal pain . Clinical examination revealed signs of toxemia and acute abdomen [26].

Gibney and Gulati reported cholecystitis, pneumonia , gastrointestinal bleed,intestinal perforation and osteomyelitis in patients with typhoid perforation [28,30]. Perforation was commonly seen to occur in the second week of onset of illness[17,18,24,25] . Keenan et al reported that 88% of his patients perforated in the second week of illness [17] . Lizzaralde et al has reports that 54.2% of his patients perforated in the second week [25] .

Chest X-ray is a gold standard investigation to detect hollow viscus perforation. Free gas under the diaphragm was seen in about 75.7% of perforations and in 94% 0f blunt injury perforation. Abdominal X-ray revealed features suggestive of ileus.

Pneumoperitoneum has been reported in 52% to 82% of cases in studies by Hadley, Archampong, Vaidyanathan ,and Tacyildiz [17,19,22,48] .

Positive Widal test was reported in 30% of typhoid perforation patients by Kaul and in 46.1% of his patients by Santillana [18,32] . It was also reported positive in 75.5% of his cases by Jarrett and in 74% by Vaidyanathan [42 ,48]. Four-fold increase of titres is considered more significant [34] .

Hadley reported positive cultures in 22% and Santillana in 47% of patients [17,18] . Prior use of antibiotic therapy was probably the reason for the low isolation the study [17 ,19 ,34] . Another cause would be a delay in plating the samples.

Tuberculosis was diagnosed definitively by histopathology . The presence of Erythrophagocytosis ( RBC s within the aggregates ) virtually confirms the diagnosis of typhoid perforation [29].

Though all the tests are supplementary in the diagnosis of typhoid, Widal is the most useful of the all . It is easily available in labs and is less susceptible to prior antibiotic therapy when compared with blood culture. This usefulness was also confirmed by Jarrett [40] .

In this study one patient with confirmed typhoid was treated with ciprofloxacin and metronidazole.

In the management of typhoid perforation some authors advocated conservative line of management [36,37,38] . Currently there no such controversies exist in the treatment of typhoid perforation with the present recommendation being surgical management [20] . The various surgical options in use are local drainage, simple two layer closure, closure with an

omental patch, wedge resection, resection- anastomosis, ileo transverse anastomosis and ileostomy [25,28,32,40,41,42] .

In this study patients underwent either simple double layer closure, omental patch closure or resection anastomosis. Nil patients were treated by conservative measures, wedge resection, ileostomy or ileotransverse anastomosis . Resection anastomosis was employed in typhoid and traumatic perforations where multiple perforations were found on laparotomy.

Orloff recommended debridement and closure in traumatic perforation cases when the injury was small and advocated resection anastomosis in patients with larger wounds or multiple perforations [56] .

The overall complication rate for all patients in this series was 78%. In accordance with the literature reports typhoid perforations are associated with a high morbidity rates between 28.5% and 81% [17,18,22,24,26] . Santillana in his case series reported a overall morbidity rate of 71.9% in 96 patients.

The common complications encountered were wound infection, wound dehiscence, pelvic abscess, fecal fistula and respiratory complications which compare with published reports [17,18,21,23] . Wound infections accounted for about 61.4% of operated patients . Anastomotic leak was seen in 4.2% of these patients .

Literature reports a rate of leak between 3% and10% [17,18,21] . The high faecal fistula rate in literatures are due to later presentation of patients, since most of their patients presented late. Ihekwaba and Shittu recommends early closure of fistula in developing countries because of poor resources [49] . According to studies the mortality rate of the patients with fistula is improved with both total parenteral nutrition and better antibiotic cover.

Patients with traumatic perforations had comparitively lesser complications possibly due to a healthier bowel than those of patients with typhoid or non-specific perforations. In patients with traumatic perforations outcome is primarily influenced by injury to other solid organs [56].

The mortality in this series was 5.7%. 2.9% of patients of traumatic perforation expired . Nil patient died in penetrating injury group . The morality rate in non specific perforation was 16.6 % . This difference should pose a trend towards significance on statistical analysis.

The surgical procedure had not influenced either the morbidity or the mortality in patiens irrespective of their etiology . Resection anastomosis was found to have a higher mortality rate of about 7.7% .

Eggleston reported that the surgical procedure done did not influence the outcome [26] . Talwar and Sharma reported the least mortality rate with early primary closure and Ameh et al found highest mortality rate with wedge resection and least with resection anastomosis [41,42] .

Lag period has been shown to influence both the mortality and morbidity. Regression analysis showed that the mortality and morbidity rates were found to be increased with increasing lag period. This association was also found in patients with typhoid perforations though only a trend was seen in patients with non specific perforations. Increasing lag period was found to be associated with increased mortality rates in case series by Archampong, Eggleston, Bose and Talwar [19,26,41,51] .

In patients of ileal perforation the factors significantly influencing the mortality rates are age greater than 50yrs, female sex, faeculent peritonitis, raised blood urea and creatinine as per the Manheim's peritonitis index. In this study age > 50 yrs , shock at presentation and lag period of >48 hrs were significant factors influencing the mortality. Increasing trends were seen with faecal fistula formation , etiology of typhoid and the preoperative azotemia. Sex, hemoglobin levels and albumin values, number of

perforations and the type of peritoneal contamination were not found to be significant factors .

Archampong reports points out that urine output prior to surgey, serum Potassium and blood urea significantly affected survival in patients with typhoid perforation.

Mock et al reported that multiple perforations, generalised contamination of the peritoneal cavity and single layer closure of the perforation influenced the survival greatly [(21)].

Eggleston in his case series of 78 patients advocated that the shock, uraemia, encephalopathy, faecal peritonitis and postoperative faecal fistula as strong predictors of mortality [(26)] .

# *SUMMARY*
# *&*
# *CONCLUSIONS*

# SUMMARY AND CONCLUSIONS

This study was conducted from October 2010 to October 2012. It includes seventy cases of ileal perforation admitted in Mahatma Gandhi Memorial Government Hospital, Trichy in that period. Etiology, presentation, management and outcome of patients with ileal perforations were studied with emphasis on traumatic, non-specific, tubercular and typhoid perforations and the factors that influenced the prognosis.

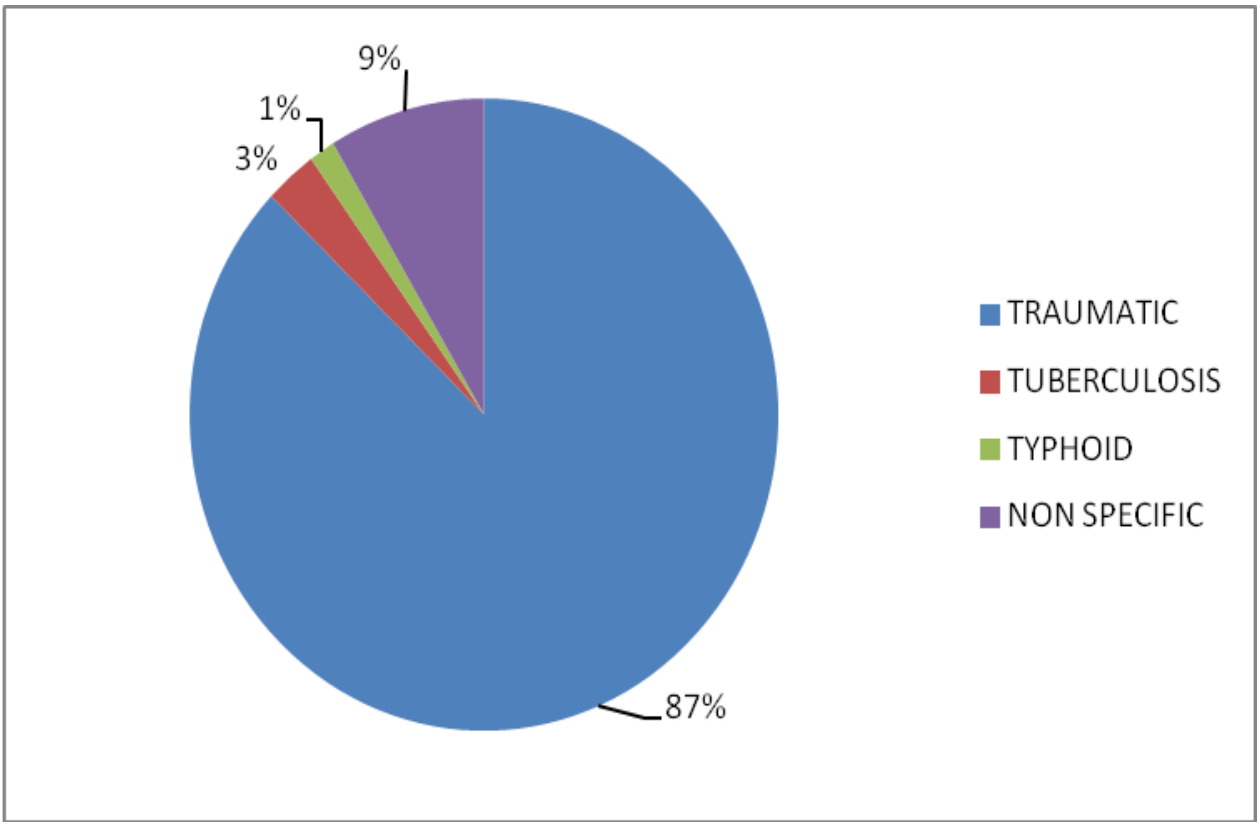
- Trauma is the most common cause of Ileal perforation, followed by non-specific perforations.
- Patients have a male preponderance and are usually in the third and fourth decades of their lives.
- All patients of Ileal perforation presented with abdominal pain.
- Many patients presented with guarding and rigidity , more than half of the patients with perforation due to penetrating abdomen presented with soft abdomen.
- X-Ray Chest PA view and Abdomen Erect view is a useful tool in the diagnosis of Ileal Perforation.

- Many patients were taken up for surgery within 24hrs of onset of pain.
- Single perforation was seen in many traumatic patients and non-specific , tubercular and typhoid cases presented with multiple perforations.
- Perforation was identified within 30 cm of Ileocaecal junction in many of the blunt injury patients, whereas in penetrating injury the site of perforation was proximal ileum in many cases
- Many patients underwent 2-layer closure , followed by resection anastamosis .
- Most of the patients developed complications. Wound infection , the commonest followed by pelvic abscess and respiratory complication.
- The type of surgical procedure did not influence outcome, either morbidity or mortality.
- Lag period significantly influenced outcome. This was true for cases of ileal perforation irrespective of etiology .

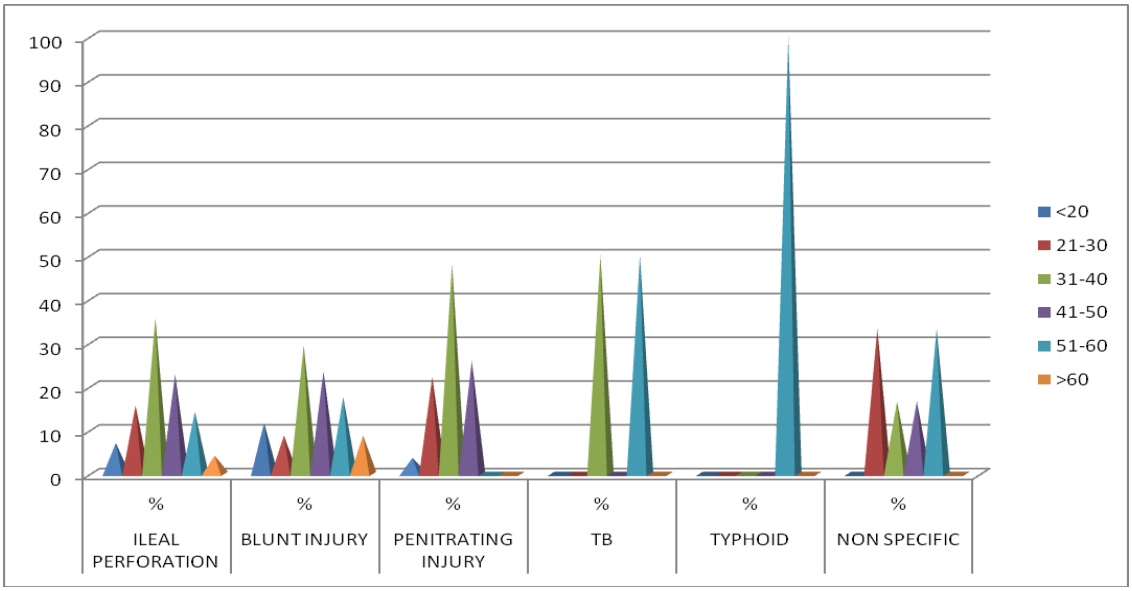
- Morbidity and Moratality was significantly influenced by age greater than 50, fecal peritonitis ,and shock.
- Only few patients developed anastamotic leak which shown good healing nature of ileum, irrespective of type of surgery
- Perforation due to typhoid was significantly reduced due to early diagnosis with rapid investigations which has more sensitivity and specificity and better treatment with antibiotics .

# *FIGURES*

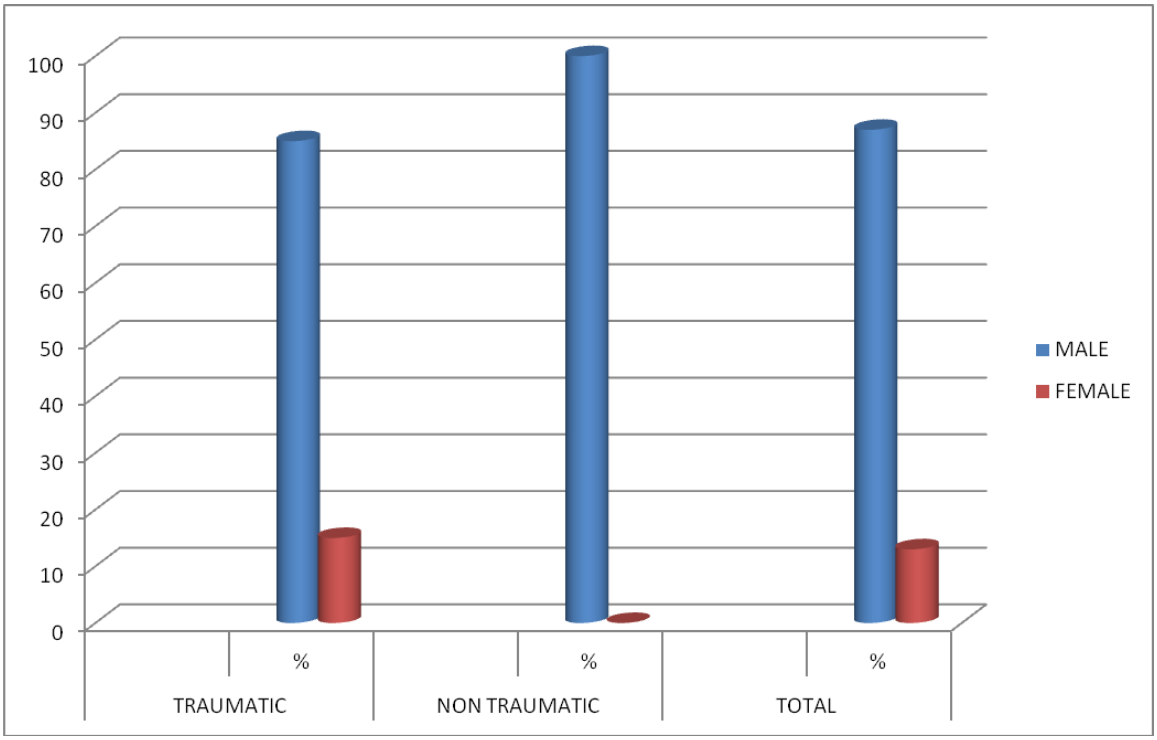
**CAUSES OF ILEAL PERFORATION**



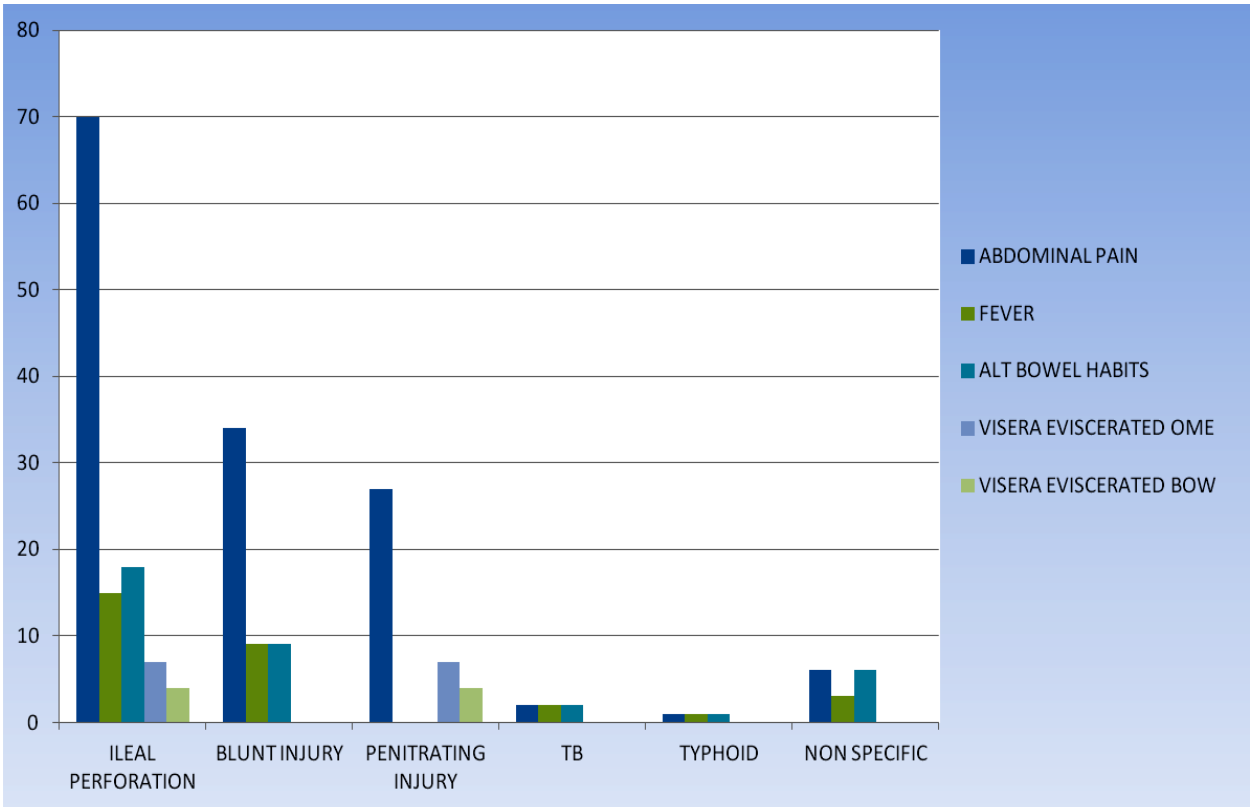
## AGE INCIDENCE IN ILEAL PERFORATION



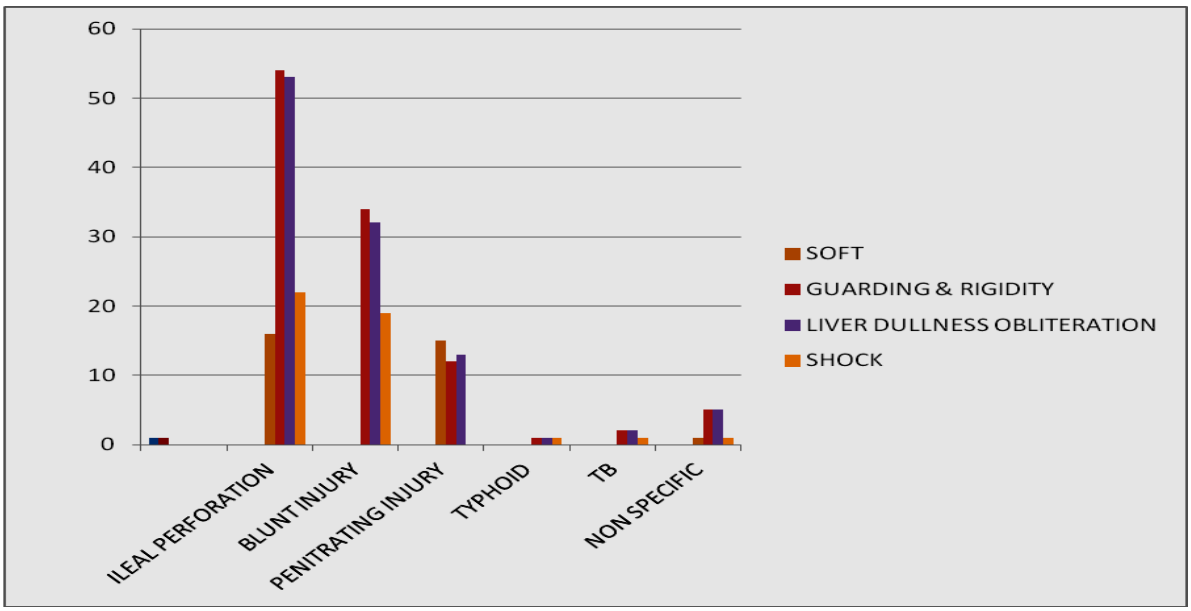

## SEX INCIDENCE IN ILEAL PERFORATION

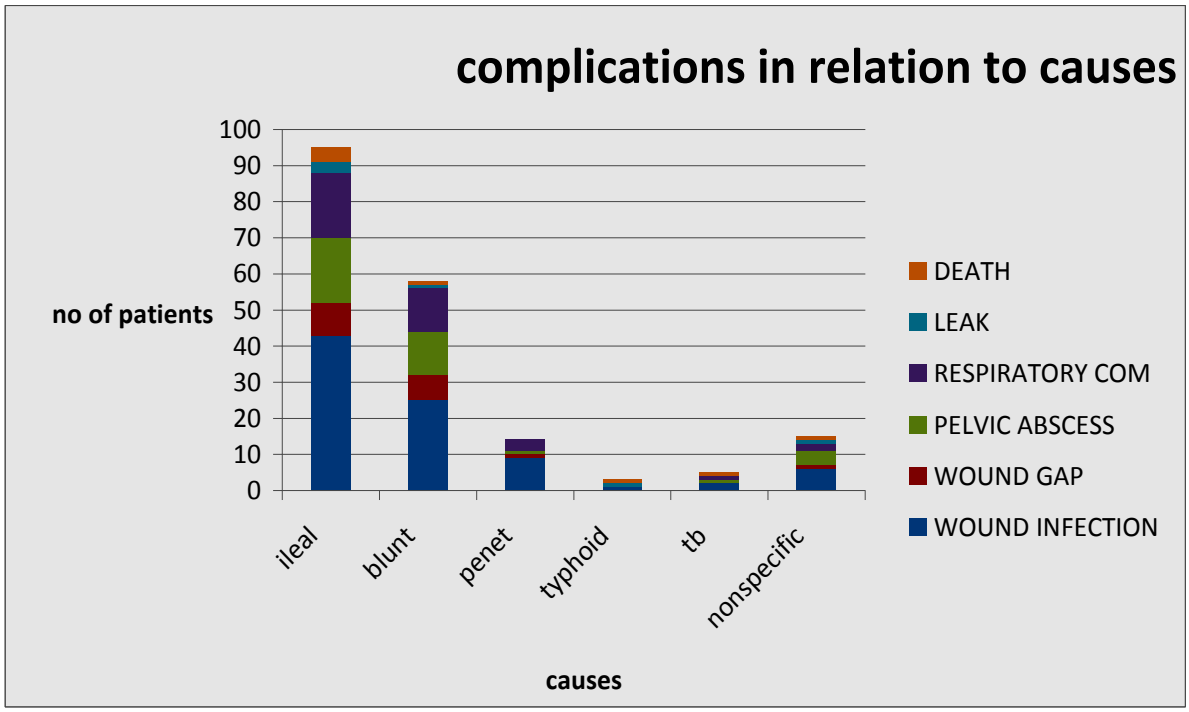


## SYMPTOMS IN ILEAL PERFORATION

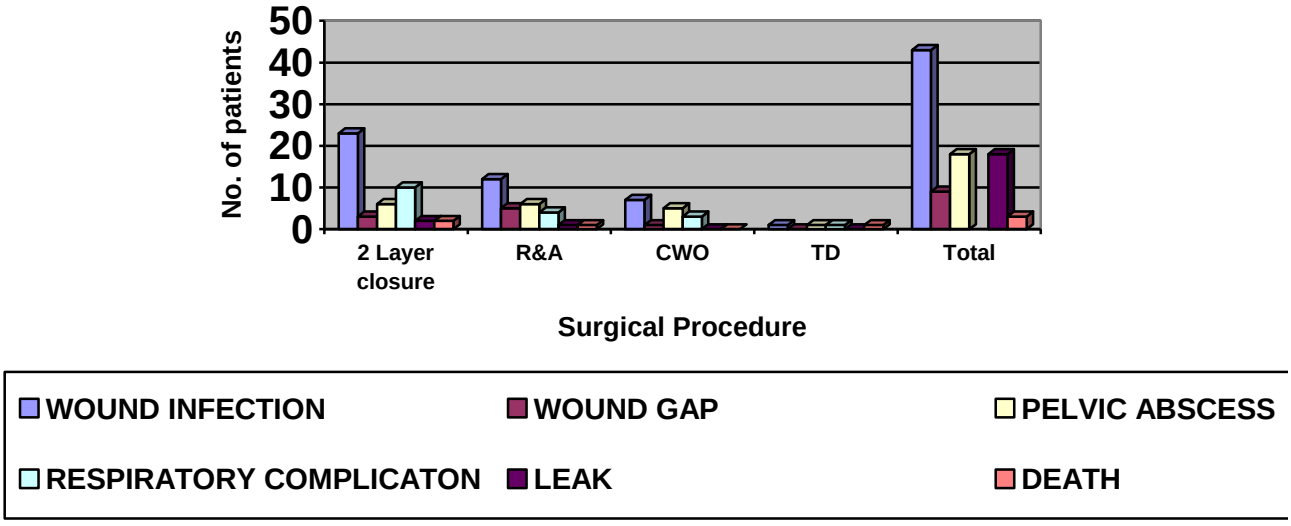



## SIGNS IN ILEAL PERFORATION

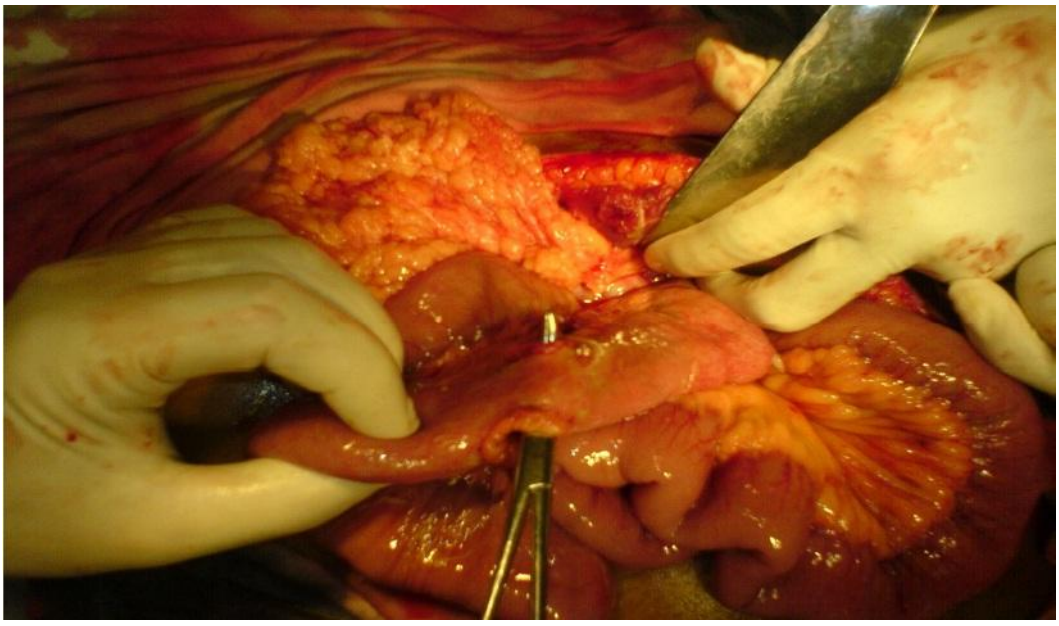





**Complication in Relation to Surgical Procedure**



**STAB INJURY INVOLVING SMALL BOWEL**

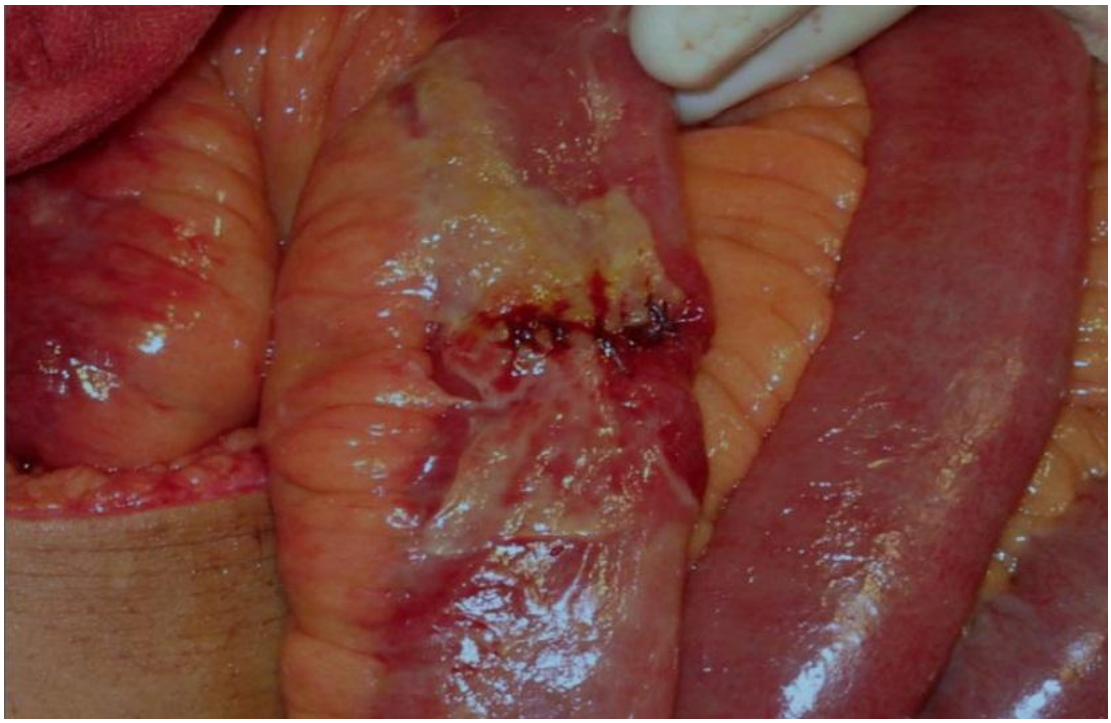


**STAB INJURY WITH OMENTAL PROLAPSE**

## PENETRATING INJURY

## WOUND SITE INFECTION AND GAPING

## SIMPLE TWO LAYER CLOSURE



## RESECTION AND ANASTAMOSIS

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# *ANNEXURE*

# A COMPREHENSIVE STUDY ON ILEAL PERFORATION

## PROFORMA

Name: Age/Sex: IP No:

Date & Time of ADMISSION :

Date & Time of SURGERY :

**Presenting Complaints:**

| Abdominal pain | Distension | Bowel habits | Fever | Viscera Evisceration | Others |
|---|---|---|---|---|---|
| | | | | | |

History:

*If injury* , mode of injury-

time of injury-

Haemodynamic status : Stable/unstable

Per Abdomen findings:

| Soft | Guarding rigidity | Liver Dullness obliteration | Viscera Eviscerated | Others |
|---|---|---|---|---|
| | | | | |

Details of Extra-Abdominal Injuries if any:

**Investigations**

Air under Diaphragm in X-ray : Yes / No

Blood investigations :

USG Findings if done :

4-Quadrant Aspiration : Normal/Peritoneal Fluid/Blood

**Others:**

**Pre-op Diagnosis:**

**Per-op Findings:**

| **No.** | **Site** | **Size** | **Peritoneal Fluid** |
|---|---|---|---|
| | | | |

**Associated Findings:**

**Procedure Done:** Primary Closure / Resection Anastamosis / Closure with Omental Patch/ Other

**Post OP Complications and its management:**

| **Complication** | **Wound Infection** | **Wound Gaping** | **Respiratory complication** | **Pelvic Abscess** | **Death** | **Others** |
|---|---|---|---|---|---|---|
| | | | | | | |
| Treatment given | | | | | | |

**Day of Discharge :**

**Significant Details if any:**

# MASTER CHART

| Sl. No. | Name | IP No. | Age | Sex | Clinical Features | | | | | | | | AUD | Diagnosis | Lag period | Operative findings | | | | | Procedure done | Post op Complications | | | | | Stay |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | ABDP | Fev. | B. H | Vis. Evi. | Soft | G & R | LDO | Sck | | | | No | Site. | Size | PF | AF | | WI | WG | ABS | RC | Lk | |
| 1 | Arunkrishnan | 56723 | 16 | M | + | - | - | - | - | + | + | + | + | BI | 12 hrs | 1 | 40 | 1 | BL | LI | 2 LC | - | - | - | - | - | 16 days |
| 2 | Karuppiah | 22904 | 55 | M | + | - | - | - | - | + | - | - | - | BI | 14 hrs | 1 | 20 | 0.5 | BL | MI | 2 LC | + | + | + | - | - | 20 days |
| 3 | Kannan | 23496 | 19 | M | + | - | - | - | - | + | + | - | + | BI | 12 hrs | 4 | 40 | 1 | - | - | R & A | - | - | - | - | - | 16 days |
| 4 | Ganapathy | 27252 | 18 | M | + | - | - | - | - | + | + | - | + | BI | 10 hrs | 1 | 20 | 2 | - | - | 2 LC | + | + | - | - | - | 20 days |
| 5 | Anand | 30093 | 38 | M | + | - | - | - | - | + | + | - | + | BI | 16 hrs | 4 | 30 | <2 | - | - | R & A | + | - | + | + | - | 22 days |
| 6 | Rengasamy | 46731 | 60 | M | + | + | CON | - | - | + | + | + | + | BI | >48 hrs | 1 | 20 | 2 | FAE | - | 2 LC | + | - | + | + | + | 14 days |
| 7 | Palanisamy | 52121 | 45 | M | + | - | - | - | - | + | - | + | - | BI | 24 hrs | 4 | 40 | <2 | BL | MI | R & A | + | - | - | + | - | 20 days |
| 8 | Sakthivel | 24821 | 35 | M | + | - | - | - | - | + | + | - | + | BI | 12 hrs | 1 | 20 | 1 | - | - | 2 LC | - | - | - | - | - | 14 days |
| 9 | Rajangam | 37680 | 45 | M | + | + | CON | - | - | + | + | + | + | BI | 72 hrs | 1 | 25 | 0.5 | FAE | - | CWO | + | - | + | + | - | 20 days |
| 10 | Ramasamy | 40836 | 60 | M | + | + | CON | - | - | + | + | + | + | BI | 60 hrs | 1 | 30 | 1 | FAE | - | CWO | + | + | + | - | - | 22 days |
| 11 | Periyanayagi | 58156 | 60 | F | + | - | - | - | - | + | + | - | + | BI | 12 hrs | 1 | 20 | 1 | - | - | 2 LC | + | - | - | + | - | 20 days |
| 12 | Muthulakshmi | 59812 | 45 | F | + | - | - | - | - | + | + | - | + | BI | 12 hrs | 1 | 10 | 1 | FAE | - | 2 LC | + | - | + | + | - | 18 days |
| 13 | Sekar | 10290 | 40 | M | + | - | - | - | - | + | + | - | + | BI | 8 hrs | 1 | 50 | 1 | BI | MI | 2 LC | + | - | - | - | - | 14 days |
| 14 | Anbarasan | 11214 | 36 | M | + | - | - | - | - | + | + | + | + | BI | 12 hrs | 4 | 30 | <2 | BI | MI | R & A | + | - | - | - | - | 14 days |
| 15 | Kaliyaperumal | 11821 | 50 | M | + | - | - | - | - | + | + | - | + | BI | 14 hrs | 2 | 15 | <1 | - | - | 2 LC | + | - | - | + | - | 16 days |
| 16 | Rajeswari | 16214 | 35 | F | + | - | - | - | - | + | + | - | + | BI | 12 hrs | 1 | 10 | 0.5 | - | - | 2 LC | - | - | - | - | - | 10 days |
| 17 | Nagarajan | 20065 | 60 | M | + | - | - | - | - | + | + | + | + | BI | 72 hrs | 1 | 15 | 2 | FP | - | CWO | + | - | + | + | - | 20 days |
| 18 | Avudainayagi | 59216 | 45 | F | + | - | - | - | - | + | + | + | + | BI | 14 hrs | 1 | 10 | 1 | BL | LI MI | 2 LC | + | - | - | - | - | 14 days |
| 19 | Sivakumar | 52300 | 24 | M | + | - | - | - | - | + | + | + | + | BI | 36 hrs | 1 | 20 | 1 | - | - | 2 LC | + | - | - | - | - | 16 days |
| 20 | Jeganathan | 50026 | 35 | M | + | - | - | - | - | + | + | + | + | BI | 12 hrs | 1 | 15 | 0.5 | BL | - | 2 LC | - | - | - | - | - | 14 days |
| 21 | Sappani | 60632 | 50 | M | + | + | CON | - | - | + | + | + | + | BI | 50 hrs | 1 | 30 | 1.5 | FAE | - | R & A | + | + | + | + | - | 24 days |
| 22 | Ponnusamy | 17078 | 40 | M | + | - | - | - | - | + | + | - | + | BI | 12 hrs | 1 | 25 | 1 | - | - | 2 LC | - | - | - | + | - | 16 days |

| Sl. No. | Name | IP No. | Age | Sex | Clinical Features | | | | | | | | AUD | Diagnosis | Lag period | Operative findings | | | | | Procedure done | Post op Complications | | | | | Stay |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | ABDP | Fev. | B. H | Vis. Evi. | Soft | G & R | LDO | Sck | | | | No | Site. | Size | PF | AF | | WI | WG | ABS | RC | Lk | |
| 23 | Samykannu | 44712 | 62 | M | + | + | CON | - | - | + | + | + | + | BI | 50 hrs | 1 | 10 | 1 | FAE | - | 2 LC | + | - | + | - | - | 16 days |
| 24 | Duraisamy | 17687 | 45 | M | + | - | - | - | - | + | + | - | + | BI | 8 hrs | 1 | 10 | 1 | - | - | 2 LC | + | - | - | - | - | 14 days |
| 25 | Anumandhan | 27943 | 65 | M | + | - | - | - | - | + | + | + | + | BI | 10 hrs | 1 | 5 | 2 | BL | SI | 2 LC | + | - | + | - | - | 16 days |
| 26 | Ganapathy | 28984 | 22 | M | + | - | - | - | - | + | + | - | + | BI | 8 hrs | 1 | 45 | 3 | - | - | 2 LC | - | - | - | - | - | 12 days |
| 27 | Kaliappan | 36512 | 30 | M | + | - | - | - | - | + | + | + | + | BI | 12 hrs | 1 | 10 | 2 | LI | BL | 2 LC | - | - | - | - | - | 14 days |
| 28 | Murali | 38165 | 32 | M | + | - | - | - | - | + | + | - | + | BI | 12 hrs | 1 | 10 | 1 | - | - | 2 LC | + | - | - | - | - | 12 days |
| 29 | Anthony Das | 41567 | 60 | M | + | + | CON | - | - | + | + | + | + | BI | >48 hrs | 1 | 5 | 1 | - | - | CWO | + | - | - | - | - | 16 days |
| 30 | Masanam | 55213 | 40 | M | + | - | - | - | - | + | + | + | + | BI | 14 hrs | 3 | 20 | <3.5 | BL | MI | R & A | + | + | + | - | - | 24 days |
| 31 | Nagarathinam | 55395 | 65 | M | + | + | CON | - | - | + | + | - | + | BI | >72 hrs | 1 | 10 | 1 | PF | - | CWO | + | - | + | + | - | 24 days |
| 32 | Selvi | 1631 | 45 | F | + | - | - | - | - | + | + | + | + | BI | 12 hrs | 5 | 15 | <1 | - | - | R & A | + | + | - | - | - | 24 days |
| 33 | Mohammed Hussa | 1851 | 18 | M | + | - | - | - | - | + | + | - | + | BI | 12 hrs | 1 | 10 | 2 | PF | - | 2 LC | - | - | - | - | - | 10 days |
| 34 | Velankanni | 12521 | 40 | F | + | + | CON | - | - | + | + | + | + | BI | >48hrs | 1 | 10 | 1 | PF | - | 2 LC | + | + | - | + | - | 24 days |
| 35 | Ganeshmoorthy | 51317 | 38 | M | + | - | - | OM | + | - | - | - | - | PI | 6 hrs | 1 | 90 | 0.5 | - | OI | 2 LC | - | - | - | - | - | 12 days |
| 36 | Thalapathy | 138 | 29 | M | + | - | - | BO | + | - | + | - | + | PI | 8 hrs | 1 | 80 | 1 | - | MI | 2 LC | - | - | - | - | - | 16 days |
| 37 | Baskar | 8294 | 30 | M | + | - | - | - | - | + | + | - | + | PI | 12 hrs | 2 | 70 | <1 | - | CI | 2 LC | - | - | - | - | - | 18 days |
| 38 | Neethipathy | 44110 | 23 | M | + | - | - | - | - | + | + | - | + | PI | 14 hrs | 1 | 60 | 2 | - | - | 2 LC | - | - | - | - | - | 16 days |
| 39 | Stalin | 57134 | 18 | M | + | - | - | OM | + | - | - | - | - | PI | 12 hrs | 1 | 30 | 1.5 | - | OI | 2 LC | - | - | - | - | - | 14 days |
| 40 | Manickam | 59686 | 40 | M | + | - | - | - | + | - | - | - | - | PI | 14 hrs | 1 | 30 | 0.5 | - | - | 2 LC | - | - | - | - | - | 14 days |
| 41 | Natesan | 10459 | 35 | M | + | - | - | - | + | - | - | - | - | PI | 10 hrs | 1 | 70 | 0.5 | - | - | 2 LC | + | - | - | - | - | 16 days |
| 42 | Avudaiyappan | 12156 | 45 | M | + | - | - | OM | + | - | - | - | - | PI | 10 hrs | 1 | 60 | 0.5 | - | OI | 2 LC | - | - | - | - | - | 12 days |
| 43 | Murugan | 58164 | 22 | M | + | - | - | - | + | - | - | - | - | PI | 24 hrs | 1 | 70 | 0.5 | - | - | 2 LC | - | - | - | - | - | 10 days |
| 44 | Palani | 60251 | 35 | M | + | - | - | BO | - | + | + | - | + | PI | 8 hrs | 2 | 70 | <1.5 | - | MI | 2 LC | + | - | - | - | - | 16 days |
| 45 | Sankar | 61815 | 45 | M | + | - | - | - | + | - | - | - | - | PI | 8 hrs | 1 | 70 | 0.5 | - | - | 2 LC | - | - | - | + | - | 16 days |
| 46 | Arokyasamy | 25232 | 45 | M | + | - | - | OM | - | + | + | - | + | PI | 12 hrs | 3 | 45 | <1 | - | MI | R & A | + | + | - | - | - | 16 days |

| Sl. No. | Name | IP No. | Age | Sex | Clinical Features | | | | | | | | AUD | Diagnosis | Lag period | Operative findings | | | | | Procedure done | Post op Complications | | | | | Stay |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | ABDP | Fev. | B. H | Vis. Evi. | Soft | G & R | LDO | Sck | | | | No | Site. | Size | PF | AF | | WI | WG | ABS | RC | Lk | |
| 47 | Alagarsamy | 28774 | 33 | M | + | - | - | - | - | + | + | - | + | PI | 8 hrs | 1 | 60 | 0.5 | - | - | 2 LC | + | - | - | - | - | 18 days |
| 48 | Kumar | 29258 | 35 | M | + | - | - | - | - | + | + | - | + | PI | 10 hrs | 1 | 80 | 1 | - | MI | 2 LC | - | - | - | - | - | 10 days |
| 49 | Arokyam | 37394 | 47 | F | + | - | - | OM | + | - | - | - | - | PI | 6 hrs | 1 | 90 | 0.5 | - | MI | 2 LC | - | - | - | + | - | 14 days |
| 50 | Sankar | 57977 | 39 | M | + | - | - | - | - | + | + | - | + | PI | 8 hrs | 2 | 90 | <2 | BI | - | 2 LC | - | - | - | - | - | 12 days |
| 51 | Yuvaraj | 8289 | 40 | M | + | - | - | OM | - | + | + | - | + | PI | 8 hrs | 2 | 80 | <1.5 | - | OI | 2 LC | - | - | - | - | - | 12 days |
| 52 | Krishna | 18871 | 22 | M | + | - | - | BO | - | + | + | - | + | PI | 6 hrs | 1 | 80 | 1 | - | CI | 2 LC | - | - | - | - | - | 14 days |
| 53 | Raja | 29965 | 35 | M | + | - | - | - | + | - | - | - | - | PI | 24 hrs | 1 | 90 | 0.5 | - | - | 2 LC | - | - | - | - | - | 10 days |
| 54 | Manickam | 30095 | 40 | M | + | - | - | - | + | - | - | - | - | PI | 36 hrs | 1 | 80 | 0.5 | - | - | 2 LC | - | - | - | - | - | 10 days |
| 55 | Kalavathy | 30112 | 45 | F | + | - | - | - | + | - | - | - | - | PI | 24 hrs | 1 | 70 | 0.5 | - | - | 2 LC | + | - | - | - | - | 12 days |
| 56 | Nagarajan | 37213 | 22 | M | + | + | CON | - | + | - | - | - | - | PI | 24 hrs | 1 | 60 | 0.5 | - | - | 2 LC | - | - | - | - | - | 12 days |
| 57 | Saroja | 49235 | 50 | F | + | - | - | OM | - | + | + | - | + | PI | 12 hrs | 1 | 45 | 2.5 | - | OI | 2 LC | + | - | - | - | - | 16 days |
| 58 | Tamizhselvan | 51372 | 35 | M | + | - | - | - | - | + | + | - | + | PI | 14 hrs | 4 | 30 | <2.5 | - | OI | R & A | + | - | + | - | - | 20 days |
| 59 | Paul pandi | 52511 | 50 | M | + | - | - | - | + | - | - | - | - | PI | 24 hrs | 1 | 70 | 0.5 | - | - | 2 LC | + | - | - | - | - | 12 days |
| 60 | Arunachalam | 54321 | 40 | M | + | - | - | BO | - | + | + | - | + | PI | 12 hrs | 2 | 60 | <1 | - | - | 2 LC | - | - | - | + | - | 20 days |
| 61 | Shanmugaraj | 11231 | 32 | M | + | - | - | - | + | - | - | - | - | PI | 36 hrs | 1 | 80 | 0.5 | - | - | 2 LC | + | - | - | - | - | 10 days |
| 62 | Velmurugan | 54709 | 40 | M | + | + | ALT | - | - | + | + | + | + | TB | >48hrs | 3 | 30 | <1.5 | FAE | ADH | TU.DR | + | - | + | + | - | Death |
| 63 | Raju | 16482 | 60 | M | + | + | CON | - | - | + | + | + | + | TB | >48hrs | 2 | 10 | <1 | FAE | NOD | CWO | + | - | - | - | - | 20 days |
| 64 | Rajesh kumar | 8421 | 55 | M | + | + | CON | - | - | + | + | + | + | TY | >72hrs | 2 | 20 | <1 | FAE | - | 2 LC | + | - | - | - | + | Death |
| 65 | Padmanaban | 3188 | 60 | M | + | - | ALT | - | + | - | - | - | - | NS | >72hrs | 3 | 40 | <1.5 | FAE | - | R & A | + | - | - | - | + | Death |
| 66 | Mohanraj | 42453 | 23 | M | + | - | ALT | - | - | + | + | - | + | NS | >48hrs | 1 | 30 | 2 | FAE | - | CWO | + | - | + | - | - | 20 days |
| 67 | Vijayakumar | 28943 | 27 | M | + | + | CON | - | - | + | + | - | + | NS | 72 hrs | 1 | 30 | 2 | - | STR | R & A | + | + | + | + | - | 24 days |
| 68 | Abdul sameer | 84907 | 52 | M | + | + | CON | - | - | + | + | + | + | NS | 48 hrs | 2 | 20 | 1 | - | DIV | R & A | + | - | + | - | - | 20 days |
| 69 | Manickavasagam | 19821 | 40 | M | + | - | CON | - | - | + | + | - | + | NS | 40 hrs | 2 | 15 | 0.5 | FAE | BAN | R & A | + | - | - | - | - | 20 days |
| 70 | Gokul Das | 24143 | 41 | M | + | + | CON | - | - | + | + | - | + | NS | 72 hrs | 2 | 15 | <1 | FP | IA | 2 LC | + | - | + | + | - | 24 days |

# LIST OF ABBREVIATIONS USED IN MASTER CHART

M -Male

F- Female

ABD P - Abdominal Pain

Fev-Fever

B.H-Bowel Habits

ALT-altered

CON- Constipation

Vis.Evi- Viscera Eviserated

OM- Omentum

BO-Bowel

G&R- Guarding and Rigidity

LDO- Liver Dullness Obliteration

SCK-Shock

AUD-Air Under Diaphragm

BI- Blunt Injury

PI-Penetrating Injury

TB-Tuberculosis

TY-Typhoid

NS-Non Specific

BL- Blood

PF- Peritoneal Fluid

SITE-Distance of perforation site from Ileo-Caecal Junction

FAE- Faecal

FP- Faeco-Purulent

MI-Mesentric Injury

OI-Omental Injury

LI- Liver Injury

CI- Colon Injury(Serosal Tear)

SI- splenic Injury

ADH-Adhesion

NOD- Node Enlargement

STR-Stricture

BAN- Band

DIV-Diverticulum

IA-Inflammed Appendix

2LC- Two Layer closure

R&A-Resection and Anastamosis

CWO- Closure With Omental Patch

TU DR-Tube Drainage

WI- Wound Infection

WG-Wound Gap

ABS-Pelvic Abscess

RC-Respiratory Complication

Lk-Leak from Anastamotic site